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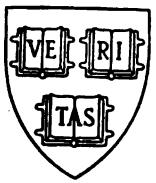


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VOLUME THE SIXTY-EIGHTH.

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## *OCTOBER*, 1885.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

*Elected*

1846 *ABERCROMBIE, JOHN, M.D.

1877 ABERCROMBIE, JOHN, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square.

1851 *ACLAND, SIR HENRY WENTWORTH, K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Broad Street, Oxford.

1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 70A, Grosvenor street.

*Elected*

1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., F.R.S., Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., F.R.S. Ed., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square, W.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's park; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Resident Surgeon, General Hospital, Nottingham.

1881 ANDERSON, JAMES, A.M., M.D., 84, Wimpole street, Cavendish square.

1862 ANDREW, EDWYN, M.D., 12, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. *Trans.* 1.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1880 *APPLETON, HENRY, M.D., Staines.

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

*Elected*

1851 *BAKER, ALFRED, Consulting Surgeon to the Birmingham General Hospital; 3, Waterloo street, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; Examiner in Surgery at the University of London; 26, Wimpole street, Cavendish square. C. 1878-9. *Lib. Com.* 1876-7. *Trans.* 6.

1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.

1848 †BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Queen's University in Ireland; 45, Merrion square, Dublin.

1879 BARKER, ARTHUR EDWARD JAMES, Surgeon to, and Assistant Professor of Clinical Surgery at, University College Hospital; 87, Harley street, Cavendish square. *Trans.* 3.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 109, Gloucester place, Portman square. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

*Elected*

1876 BARLOW, THOMAS, M.D., B.S., Physician to University College Hospital; Physician to the Hospital for Sick Children, Great Ormond street, and Assistant Physician to the London Fever Hospital; 10, Montague street, Russell square. *Trans.* 1.

1881 *BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 BARNES, ROBERT, M.D., 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1880 BARROW, A. BOYCE, Assistant Surgeon to the Westminster Hospital and to the West London Hospital; 17, Welbeck street, Cavendish square.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 55, Wimpole street, C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 10.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 20, Queen Anne street, Cavendish square. C. 1885. *Trans.* 1.

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest; and Physician to the Great Northern Hospital; 23, Upper Berkeley street.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

*Elected*

1860 *Bealey, Adam, M.D., M.A., Oak Lea, Harrogate.

1856 Beardsley, Amos, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 Beck, Marcus, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. *Referee*, 1882-5. *Lib. Com.* 1881-5.

1880 Beevor, Charles Edward, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley street, Cavendish square. *Trans.* 1.

1858 Begley, William Chapman, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877–8.

1883 Bell, Hutchinson Royes, Surgeon to, and Demonstrator of Operative Surgery at, King's College Hospital; 12, Queen Anne street, Cavendish square.

1871 Bellamy, Edward, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Lecturer on Artistic Anatomy to the Science and Art Department, South Kensington; 17, Wimpole street, Cavendish square. *Referee*, 1882-5. *Lib. Com.* 1879-81.

1847 Bennet, James Henry, M.D., The Ferns, Weybridge, and Mentone.

1880 Bennett, Alex. Hughes, M.D., Assistant Physician to the Westminster Hospital; 38, Queen Anne street, Cavendish square. *Trans.* 1.

1883 Bennett, Storer, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to the Dental Hospital of London; 17, George street, Hanover square.

1877 Bennett, William Henry, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; Surgeon to the Belgrave Hospital for Children; 1, Chesterfield street, Mayfair.

1845 †Berry, Edward Unwin, 17, Sherriff road, West Hampstead.

*Elected*

1885 BERRY, JAMES, Assistant Demonstrator of Anatomy, St. Bartholomew's Hospital; 27, Upper Bedford place.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

1854 BIRD, PETER HINCKES, F.L.S.

1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6. *Referee*, 1851-9.

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D.

1881 BISS, CECIL YATES, M.D., Assistant Physician to the Hospital for Consumption, Brompton, and to the Middlesex Hospital; 65, Harley street, Cavendish square. *Trans.* 1.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street. C. 1883-4.

*Elected*

1867 Bloxam, John Astley, Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 Bojanus, Louis Henry, M.D., Wilna.

1846 †Bostock, John Ashton, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.* 1867.

1869 Bourne, Walter, M.D. [care of the National Bank of India, 80, King William street, City]; Archaco, France.

1882 Bowlby, Anthony Alfred, Surgical Registrar to St. Bartholomew's Hospital; 75, Warrington crescent, Maida hill.

1870 *Bowles, Robert Leamon, M.D., 8, West terrace, Folkestone.

1841 †Bowman, Sir William, Bart., LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

1884 Boyd, Stanley, M.B., Assistant Surgeon to the Charing Cross Hospital; 27, Gower street.

1862 Brace, William Henry, M.D., 7, Queen's Gate terrace, Kensington.

1874 Bradshaw, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1883 Bradshaw, James Dixon, M.B., 30, George Street, Hanover square.

1867 *Brett, Alfred T., M.D., Watford, Herts.

1876 Bridges, Robert, M.B., Manor House, Yattendon, Newbury, Berks.

*Elected*

1867 BRIDGEWATER, THOMAS, M.B., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Examiner in Medicine at the University of London; 34, Seymour street, Portman square. C. 1885. *Referee*, 1881-4. *Trans.* 5.

1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

1878 BROWNE, JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; Lecturer on Mental Diseases, St. Mary's Hospital; 7, Cumberland Terrace, Regent's Park.

1880 BROWNE, JAMES WILLIAM, M.B., 8, Norland place, Uxbridge road.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.

1881 BROWNE, OSWALD A., M.A., M.B., 25, Bernard street, Russell square.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Trans.* 1.

*Elected*

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; Examiner in Materia Medica in the University of London; 50, Welbeck street, Cavendish square. *Referee*, 1880-85. *Lib. Com.* 1882-5.

1860 BRYANT, THOMAS, *Vice-President*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. V. P. 1885. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 9. *Pro.* 1.

1855 BRYANT, WALTER JOHN, Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 24, Nottingham place, Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D.

1876 BUCKNILL, JOHN CHARLES, M.D., F.R.S.; E 2, The Albany, Piccadilly, and Hill Morton Hall, Rugby.

1881 BULLER, AUDLEY CECIL, M.D., 10, Clifton hill, St. John's Wood.

1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Referee*, 1842-6, 1850-7, 1861-68, 1875-81. *Lib. Com.* 1836. *Trans.* 2.

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Referee*, 1846-54, 1857-65. *Lib. Com.* 1847. *Trans.* 4.

*Elected*

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 35, Brook street, Grosvenor square.

1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.

1871 BUTT, WILLIAM F., 48, Park street, Park lane.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer street, Cavendish square.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square. C. 1885.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1885 CAHILL, JOHN, 26, Albert Gate, Hyde park.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1863.

1879 CARTWRIGHT, S. HAMILTON, Professor of Dental Surgery at King's College; 32, Old Burlington street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. *Trans.* 1.

*Elected*

1884 CHAFFEY, WAYLAND CHARLES, M.B., Medical Registrar to the Hospital for Sick Children, Great Ormond street; 28, Cedars road, Clapham Common.

1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital and to the Lock Hospital; Shrubs Hill House, Sunningdale. C. 1861. V.P. 1867. L. 1869-72. *Referee*, 1851-60, 1866. *Lib. Com.* 1852, 1868. *Trans.* 1.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Examiner in Obstetric Medicine in the University of London; 60, Great Cumberland place. *Lib. Com.* 1885. *Trans.* 6.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; Crofton Lodge, Hopton road, Coventry park, Streatham.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple Row, Birmingham. *Trans.* 1.

1868 CHEADLE, WALTER BUTLER, M.D., Physician (with charge of out-patients) to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the Hospital for Sick Children; 19, Portman street, Portman square. *Referee*, 1885.

1879 CHEYNE, WILLIAM WATSON, M.B., Assistant Surgeon and Demonstrator of Surgery to King's College Hospital; 14, Mandeville place, Manchester square, W.

1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

*Elected*

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885. *Referee*, 1874-81.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., F.R.S., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875.

1879 CLARK, ANDREW, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 CLARKE, ERNEST, M.B., B.S., 21, Lee terrace, Blackheath.

1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1842 †CLAYTON, SIR OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1879 CLUTTON, HENRY HUGH, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 2, Portland place.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

*Elected*

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 8, Suffolk street, Pall Mall. *Trans.* 2.

1885 COLLINS, WILLIAM MAUNSELL, M.D., 10, Cadogan place.

1865 COOPER, ALFRED, Surgeon for Out-patients to the Lock Hospital; Assistant Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.P. 1873-4. *Lib. Com.* 1847, 1856-7.

1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Ormean Terrace, Belfast.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.

1860 †COUPER, JOHN, Surgeon to the London Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee* 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 14, Weymouth street, Portland place.

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.

1868 CRAWFORD, SIR THOMAS, K.C.B., M.D., Director General, Army Medical Department; 6, Whitehall yard, and 5, St. John's park, Blackheath.

1873 CREIGHTON, CHARLES, M.D., 11, New Cavendish street. *Referee*, 1882-5. *Trans.* 1.

*Elected*

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; Assistant Physician to the East London Hospital for Children; 28, Welbeck street, Cavendish square. *Trans.* 1.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. C. 1884. *Referee*, 1885. *Lib. Com.* 1877-8. *Trans.* 1.

1862 CROMPTON, SAMUEL, M.D., Brookmead, Cranleigh, Surrey.

1837 CROOKES, JOHN FARRAR, 45, Augusta gardens, Folkestone.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital; Aural Surgeon to the Great Northern Hospital; 17, Queen Anne street.

1846 CURLING, HENRY, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary; Augusta Lodge, Ramsgate, Kent.

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 27, Brunswick square, Brighton. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Referee*, 1844-6, 1851-3, 1858, 1865-70, 1875-9. *Sci. Com.* 1863. *Lib. Com.* 1839. *Trans.* 13. *Pro.* 1.

1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square. *Referee*, 1884-5.

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

*Elected*

1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 3.

1884 DALLAWAY, DENNIS, Langham Hotel.

1877 DARBISHIRE, SAMUEL DUKINFIELD, M.D., Physician to the Radcliffe Infirmary, Oxford; 60, High street Oxford.

1879 DARWIN, FRANCIS, M.B., F.R.S., The Grove, Huntingdon road, Cambridge.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 2, Marlborough buildings, Bath.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 DAVY, RICHARD, F.R.S. Ed., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square. *Trans.* 1.

1882 DAWSON, YELVERTON, M.D., Heathlands, Southbourn-on-Sea, Hants.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to St. George's Hospital; 61, Brook street. *Trans.* 2.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Senior Physician to the Hospital for Sick Children; 9, Chesterfield street, Mayfair. C. 1874-5. *Referee*, 1869-73, 1882-5. *Sci. Com.* 1867-79. *Trans.* 13.

*Elected*

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; Streate place, Bournemouth. *Trans.* 2.

1845 DODD, JOHN.

1879 DONKIN, HORATIO, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 60, Upper Berkeley street, Portman square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Assistant Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. *Trans.* 1.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley street, Cavendish square. C. 1880. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1884 DRAGE, LOVELL, St. Bartholomew's Hospital.

1879 DREWITT, F. G. DAWTREY, M.D., Assistant Physician to the West London Hospital and to the Victoria Hospital for Children; 52, Brook street, Grosvenor square.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 23, Sackville street, Piccadilly.

1865 †DUCKWORTH, DYCE, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885. *Trans.* 1.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1845 DUFF, GEORGE, M.D., High street, Elgin.

*Elected*

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

1871 DUKE, BENJAMIN, 2, Windmill road, Clapham common.

1871 *DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham common.

1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D., F.R.S., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; 71, Brook street, Grosvenor square. *Referee*, 1881-5. *Trans.* 1.

1884 DUNCAN, WILLIAM A., M.D., Assistant Obstetric Physician and Teacher of Operative Midwifery, Middlesex Hospital; 6, Harley street, Cavendish square.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., 82, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1868 EASTES, GEORGE, M.B. Lond., Surgeon-Accoucheur to the Western General Dispensary; 69, Connaught street, Hyde park square.

1883 EDMUNDS, WALTER, M.C., 79, Lambeth Palace road, Albert Embankment. *Trans.* 1.

*Elected*

1883 EDWARDES, EDWARD JOSHUA, M.D., 17, Orchard street, Portman Square, W.

1884 EDWARDS, F. SWINFORD, Surgeon to the West London Hospital; 93, Wimpole street, Cavendish square.

1824 EDWARDS, GEORGE.

1869 ELAM, CHARLES, M.D., 75, Harley street, Cavendish square.

1848 ELLIS, GEORGE VINER, late Professor of Anatomy in University College, London; Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †ERICHSEN, JOHN ERIC, LL.D.,F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C.1855-6. V.P. 1868. P.1879-80. *Referee*, 1866-7, 1884-5. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Pathological Curator of the Museum, Royal College of Surgeons; Assistant Surgeon to the London Hospital; 15, Finsbury circus. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon street, Mayfair.

1875 *FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 1, Glengall place, Belfast.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.

1862 FARQUHARSON, ROBERT, M.D., M.P., Migvie Lodge, Porchester gardens, Hyde park; Finzean, Aboyne, Aberdeenshire, and the Reform Club, Pall Mall. *Lib. Com.* 1876-80.

*Elected*

1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Referee*, 1848-54, 1861-3, 1865-6. *Sci. Com.* 1863. *Lib. Com.* 1847.

1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, late Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. *Referee*, 1881-5.

1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-5. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Forensic Medicine at King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Trans.* 2.

1852 *FIELD, ALFRED GEORGE.

1849 †FINCHAM, GEORGE TUPMAN, M.D., Consulting Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1879 FINLAY, DAVID WHITE, M.D., Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 9, Lower Berkeley street, Portman square.

1866 FISH, JOHN CROCKETT, B.A., M.D., Assistant Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1866 FITZPATRICK, THOMAS, M.D., M.A., Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.

*Elected*

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 FONMARTIN, HENRY DE, M.D., Parkhurst, Isle of Wight.

1848 †FORBES, JOHN GREGORY, Egerton House, Egerton, Ashford, Kent. C. 1868-9. *Lib. Com.* 1855. *Trans.* 2.

1852 †FORSTER, JOHN COOPER, Consulting Surgeon to the Royal Hospital for Children and Women; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V. P. 1877-8. T. 1879-84. *Referee*, 1870-2, 1876. *Pro.* 1.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 14, Temple row, Birmingham.

1883 FOWLER, JAMES KINGSTON, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to th Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician to the Skin Department of the Paddington Green Hospital for Children, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1858 FOX, WILSON, M.D., F.R.S., *Librarian*, Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. C. 1875-6. L. 1883-5. *Referee*, 1869-74. *Lib. Com.* 1866-70, 1874. *Trans.* 3.

*Elected*

1871 FRANK, PHILIP, M.D., Cannes, France.

1884 *FRANKS, KENDAL, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; 69, Fitzwilliam square, Dublin.

1843 FRASER, PATRICK, M.D. C. 1866.

1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.

1836 †FRENCH, JOHN GEORGE, 10, Cunningham place, St. John's Wood road. C. 1852-3.

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's park.

1883 FULLER, HENRY ROXBURGH, M.B., 45, Curzon street, May Fair.

1876 FURNER, WILLOUGHBY, Assistant Surgeon to the Sussex County Hospital; 2, Brunswick place, Brighton.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow.

1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant Physician to the Hospital for Sick Children; 49, Wimpole street, Cavendish square. *Referee*, 1882-5. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 45, Great Marlborough street.

1865 GANT, FREDERICK JAMES, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. C. 1880-81. *Lib. Com.* 1882-5. *Trans.* 3.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

*Elected*

1854 †GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1879 GARSTANG, THOMAS WALTER HARROPP, Dobcross, near Oldham.

1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; The Priory, Caerleon, Monmouthshire. C. 1875-6. *Trans.* 2.

1819 GAULTER, HENRY.

1866 GEE, SAMUEL JONES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children; 54, Wimpole street, Cavendish square. C. 1883-4. *Sci. Com.* 1879. *Referee*, 1885. *Lib. Com.* 1871-6. *Trans.* 1.

1878 GERVIS, HENRY, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; 40, Harley street, Cavendish square. *Referee*, 1884-5.

1884 GIBBES, HENEAGE, M.D., Physician to the Metropolitan Dispensary; Lecturer on Morbid Histology, Westminster Hospital; 44, Charleville road, West Kensington.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 32, Cadogan place.

1877 GODLEE, RICKMAN JOHN, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London; Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Consumption, Brompton; 81, Wimpole street, Cavendish square. *Trans.* 1.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

*Elected*

1883 GOODHART, JAMES FREDERIC, M.D., Assistant Physician to, and Curator of the Museum at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; Surgeon to the North-west London Hospital; 16, Queen Anne street, Cavendish square. *Trans.* 1.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine in University College, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. *Lib. Com.* 1884-5. *Trans.* 6.

1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Mixbury, Eastbourne, Sussex. C. 1863.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. *Referee*, 1882-5.

1875 *GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 35, Cavendish square. C. 1871-2. *Referee*, 1876-7. *Trans.* 1.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; Castle Lodge, Reigate. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.

*Elected*

1882 GRESSWELL, DAN ASTLEY, M.B., 87, Queen's crescent, Haverstock hill.

1884 GRIFFITHS, HERBERT TYRRELL, M.D., 57, Brook street.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; 27, Curzon street, Mayfair.

1852 GROVE, JOHN, Fyning, Austen road, Guildford.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

1849 †GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Referee*, 1855-63. *Trans.* 4.

1885 GULLIVER, GEORGE, M.B., Assistant Physician to, and Lecturer on Comparative Anatomy at, St. Thomas's Hospital; 16, Welbeck street.

1883 GUNN, ROBERT MARCUS, M.B., 54, Queen Anne street, Cavendish square.

1854 †HABERSHON, SAMUEL OSBORNE, M.D., 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. V.P. 1881-2. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.

1885 HAIG, ALEXANDER, M.B., 30, Welbeck street, Cavendish square.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Forensic Medicine at the Westminster Hospital; Physician to St. Mark's Hospital; 46, Queen Anne street, Cavendish square.

1885 HALLIBURTON, WILLIAM DOBINSON, M.D., 135, Gower street.

1870 HAMILTON, ROBERT, Surgeon to the Royal Southern Hospital, Liverpool; 1 Prince's road, Liverpool.

*Elected*

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.

1856 †HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and late Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4.

1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-5. *Sci. Com.* 1879. *Trans.* 9.

1880 HARRIS, VINCENT DORMER, M.D., Assistant Physician to the Victoria Park Hospital; Demonstrator of Physiology at St. Bartholomew's Hospital; 39, Wimpole street, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Deputy Surgeon-General, Madras Army (retired); late Professor of Midwifery and Diseases of Women and Children, Madras Medical College.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool. *Trans.* 1.

1854 HAVILAND, ALFRED.

1870 HAWARD, J. WARRINGTON, Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 16, Savile row, Burlington gardens. C. 1885. *Lib. Com.* 1881–4. *Trans.* 1.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 9, Duke street, Portland place. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

*Elected*

1885 HAWKINS, FRANCIS HENRY, M.B., Physician to St. George's and St. James's Dispensary; 47, Upper Berkeley street.

1848 †HAWKSLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 65, Green street, Grosvenor square, and 20, Lewes crescent, Brighton.

1875 HAYES, THOMAS CRAWFORD, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1861 HAYWARD, WILLIAM HENRY, Corby, Grantham.

1848 *HEALE, JAMES NEWTON, M.D.

1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. *Lib. Com.* 1870-3. *Trans.* 3.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1882 HENSLEY, PHILIP J., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 7, West street, Finsbury circus. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 57, Harley street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.B., 22, Bedford square.

*Elected*

1843 †Hewett, Sir Prescott Gardner, Bart., F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Académie de Médecine," and of the "Société de Chirurgie," Paris; Chesnut Lodge, Horsham, Sussex. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-83. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

1855 Hewitt, W. M. Graily, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-85. *Lib. Com.* 1868, 1874.

1880 Hicks, Charles Cyril, M.D., Wokingham, Berks.

1873 Higgens, Charles, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1862 Hill, M. Berkeley, M.B., *Vice-President*, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 66, Wimpole street, Cavendish square. C. 1878-9. S. 1881-4. V.P. 1885. *Trans.* 1.

1867 Hill, Samuel, M.D., 22, Mecklenburgh square.

1861 *Hoffmeister, Sir William Carter, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1843 †Holden, Luther, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 Holland, Philip Alexander, M.A.

1868 Hollis, William Ainslie, M.A., M.D., Assistant-Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

*Elected*

1861 HOLMAN, WILLIAM HENRY, M.B., 68, Adelaide road, South Hampstead.

1856 HOLMES, TIMOTHY, M.A., *Treasurer*, Surgeon to St. George's Hospital; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3. V.P. 1879-80.

1846 †HOLTHOUSE, CARSTEN, 35, Essex street, Strand. C. 1863. *Referee* 1870-6. *Lib. Com.* 1859-60.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Assistant Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR ALEXANDER HADEN, Assistant Surgeon to University College Hospital, and Assistant Professor of Pathological Anatomy, University College, London; Superintendent of the Brown Institution, Wandsworth road; 80, Park street, Grosvenor Square.

1878 HOUGHTON, WALTER B., M.D., Church Villa, Warrior square, St. Leonards-on-Sea.

1865 HOWARD, BENJAMIN, M.D., New York, U.S.

1881 HOWARD, HENRY, M.B., abroad [6, The Terrace, Mount Pleasant, Cambridge].

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Examiner in Anatomy in the University of London; 10, St. Thomas's street, Southwark. *Sci. Com.* 1879. *Trans.* 2.

1884 HUGGARD, WILLIAM R., M,D., Place de la Synagogue, 2, Genève.

1857 HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-85. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 8.

*Elected*

1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 6.

1882 HUMPHRY, LAURENCE, M.B., 3, Trinity street, Cambridge.

1873 HUNTER, SIR W. GUYER, M.D., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.

1849 HUSSEY, EDWARD LAW, Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883-5. *Lib. Com.* 1864-5. *Trans.* 12. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Consulting Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., Cairo. [Athenæum Club, Pall Mall.]

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; 47, Waterloo road, south, Wolverhampton.

*Elected*

1883 JACOBSON, WALTER HAMILTON ACLAND, B.A., M.B., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 41, Finsbury square.

1825 JAMES, JOHN B., M.D.

1883 *JENKINS, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.

1851 †JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1884 JENNINGS, CHARLES EGERTON, M.S., M.B., 75, Park street, Grosvenor square.

1881 JENNINGS, WILLIAM OSCAR, M.D., 8, Rue Roy, Paris.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Royal General Dispensary; 16, Upper Wimpole street.

1883 JESSOP, WALTER HENRY H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †JOHNSON, GEORGE, M.D., F.R.S., *President*, Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib.Com.* 1860-1. *Trans.* 10. *Pro.* 1.

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall terrace, South Hampstead, and 14, Stratford place, Oxford street.

1884 JOHNSTON, JAMES, M.D., 7, Hanover square.

*Elected*

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; Gate House, Edmund street, Birmingham.

1881 JULER, HENRY EDWARD, Assistant Surgeon Royal Westminster Ophthalmic Hospital; Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street, Cavendish square.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; Surgeon to the Surgical Aid Society; 10, George street, Hanover square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene at King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Broadwater road, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1884 KESER, JEAN SAMUEL, M.D., 60, Queen Anne street.

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D., Physician to the Parell Dispensary, Bombay; Girgaum road, Bombay.

1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater.

*Elected*

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 3.

1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1885 KLEIN, EDWARD EMANUEL, M.D., F.R.S., 94, Philbeach gardens, Earl's Court.

1883 KNAPTON, GEORGE, Strathgyle, Portswood, Southampton.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865. *Referee*, 1850.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 1.

1882 LANG, WILLIAM, Ophthalmic Surgeon to the Middlesex Hospital; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885. *Lib. Com.* 1879-80.

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816 LAWRENCE, G. E.

1884 LAWSON, GEORGE, Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital; 12, Harley street.

*Elected*

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., Physician to the Paddington Green Children's Hospital; 12, Upper Berkeley street, Portman square.

1882 LEDWICH, EDWARD L'ESTRANGE, Lecturer on Surgical and Descriptive Anatomy in the Ledwich School of Medicine, Dublin; 23, Upper Leeson Street, Dublin.

1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 13. *Pro.* 2.

1884 LEE, ROBERT JAMES, M.D., Assistant Physician to the Hospital for Sick Children; 6, Savile row.

1883 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden road, Twickenham.

1869 LEGG, JOHN WICKHAM, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green street, Park lane. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1836 LEIGHTON, FREDERICK, M.D.

1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's park.

1872 *LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; 21, St. John street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital, and Demonstrator of Anatomy at St. Bartholomew's Hospital; 8, Serjeants' Inn, Fleet street.

*Elected*

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1881 LUCAS, RICHARD CLEMENT, Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.

1883 LUND, EDWARD, Professor of Surgery, Owens College; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1857 LYON, FELIX WILLIAM, M.D., 5, North Charlotte street, Edinburgh.

1882 LYONS, ISIDORE.

1867 MABERLY, GEORGE FREDERICK.

1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 15, Finsbury square. *Lib. Com.* 1882-5.

1867 MAC CORMAC, SIR WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Examiner in Surgery at the University of London; 13, Harley street. C. 1884-5. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 89, Merrion square west, Dublin. *Trans.* 2.

1880 *MACFARLANE, ALEXANDER WILLIAM, M.D., Consulting Physician to the Kilmarnock Fever Hospital and Infirmary; Walmer, Kilmarnock, N.B.

1866 MACGOWAN, ALEXANDER THORBURN, Vyvyan House, Clifton, near Bristol.

*Elected*

1880 McHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Assistant Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 22, George street, Hanover square.

1881 MACKENZIE, STEPHEN, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; Physician to the Royal London Ophthalmic Hospital; 26, Finsbury square. *Trans.* 1.

1885 MACKERN, JOHN, M.D., 30, Cambridge street, Hyde park.

1876 MACKEY, EDWARD, M.D., 1, Brunswick road, Hove, Brighton.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1876 MACNAMARA, CHARLES, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. *Referee*, 1884-5.

1881 MACREADY, JONATHAN F. C. H., Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, The Bungalow, Stoke Pogis, Bucks.

1880 MAKINS, GEORGE HENRY, St. Thomas's Hospital, Albert Embankment.

1876 MALLAM, BENJAMIN, Meadow Side, Leacroft road, Staines.

1855 MARCET, WILLIAM, M.D., F.R.S.; 39, Grosvenor street. C. 1871. *Referee*, 1866-70, 1883-5. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

*Elected*

1867 MARSH, F. HOWARD, *Secretary*, Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children, Great Ormond street; 36, Bruton street, Berkeley square. C. 1882-3. S. 1885. *Lib. Com.* 1880-1. *Trans.* 4.

1838 MARSH, THOMAS PARR, M.D.

1851 †MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. P. 1882-4. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 3.

1884 MARTIN, SIDNEY HARRIS COX, M.B., 105, Haverstock hill.

1864 MASON, FRANCIS, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. C. 1880-81. *Trans.* 1.

1883 MAUDSLEY, HENRY, M.D., Resident Medical Officer, University College Hospital, Gower street.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square. *Lib. Com.* 1875-7.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square, and 11, Montpellier row, Blackheath.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; The Limes, Bristol road, Edgbaston, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

*Elected*

1885 MILLICAN, KENNETH WILLIAM, B.A., 27, Duke street, Manchester square.

1882 MILLS, JOSEPH, 15, Henrietta street, Cavendish square.

1873 MILNER, EDWARD, Surgeon for Out-Patients to the Lock Hospital; 32, New Cavendish street, Portland place.

1883 MONEY, ANGEL, M.D., Assistant Physician to the Hospital for Sick Children, Great Ormond Street, and to the City of London Hospital for Diseases of the Chest, Victoria park; 24, Harley street. *Trans.* 2.

1873 MOORE, NORMAN, M.D., Assistant Physician and Warden of the College and Lecturer on Comparative Anatomy, Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 1.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Referee*, 1882-5. *Trans.* 9.

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of St. Mary's Hospital; 63, Montagu square.

1885 MOTT, FREDERICK WALKER, M.B., Lecturer on Physiology, Charing Cross Hospital; 55, Torrington square.

1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 6, Finsbury Circus. *Referee*, 1879-85. *Trans.* 1.

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians; Consulting Physician to the Royal Hospital for Incurables; 40, Finsbury square.

*Elected*

1873 MURRAY, IVOR, M.D., F.R.S. Ed. 8, Huntriss Row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital; 38, Weymouth street, Portland place. *Trans.* 1.

1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; Hospital, Vincent square, Westminster. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, ARTHUR THOMAS, M.D., Medical Registrar, St. George's Hospital; 24, Clarges street, Piccadilly.

1881 NALL, SAMUEL, M.B., Disley, Cheshire.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park. *Lib. Com.* 1841.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; Ophthalmic Surgeon to the Hospital for Sick Children; 5, Wimpole street, Cavendish square.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., 68, Duke street, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Mount Radford, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1884 OAKES, ARTHUR, M.D., 99, Priory road, West Hampstead.

*Elected*

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption, and Physician to the Westminster General Dispensary; 17, St. James's place.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; 13, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., Lecturer on Comparative Anatomy at the Westminster Hospital; 46, Welbeck street, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Referee*, 1864-72. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Lib. Com.* 1871-5. *Trans.* 4.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1883 *OLIVER, THOMAS, M.D., Lecturer on Practical Physiology, University of Durham; and Physician to the Newcastle-upon-Tyne Infirmary; 3, Eldon square, Newcastle-upon-Tyne.

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Silver street, Lincoln.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Examiner in Medicine at the University of London; 7, Brook street, Hanover square. *Referee*, 1884-5. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

*Elected*

1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1875 OSBORN, SAMUEL, 10, Maddox street, Regent street, and Maisonnette, Datchet, Bucks.

1879 OWEN, EDMUND, Surgeon to St. Mary's Hospital; Surgeon to the Hospital for Sick Children; 49, Seymour street, Portman square. *Trans.* 1.

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 5, Hertford street, May Fair.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon (with charge of out-patients) to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. *Referee*, 1884-5. *Trans.* 2.

1847 *PAGE, WILLIAM BOUSFIELD, Consulting Surgeon to the Cumberland Infirmary, 78, Carlisle street, Carlisle. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, Surgeon to the East London Hospital for Children; 8, Old Cavendish street. *Lib. Com.* 1885. *Trans.* 3.

1885 PARKER, RUSHTON, M.B., Assistant Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

*Elected*

1841 PARKIN, JOHN, M.D., 5, Codrington place, Brighton.

1883 PASTEUR, WILLIAM, M.D., Medical Registrar to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 19, Queen street, May Fair.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1878-85.

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Woodside, Windsor forest, Berks.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Senior Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.

1870 PERRIN, JOHN BESWICK, Vernon House, Leigh, Lancashire.

1879 *PESIKAKA, HORMASJI DOSABHAI, Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., 10, Henrietta street, Cavendish square, W.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Leinster square, Bayswater.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. *Referee*, 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

*Elected*

1841 †PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to the East London Hospital for Children; 34, Ashburn place, South Kensington.

1885 POLAND, JOHN, Demonstrator of Anatomy, Guy's Hospital; 16, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, M.D., Surgical Registrar, University College Hospital; 50, Torrington square.

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; Examiner in Forensic Medicine at the University of London; 30, Wimpole street. *Trans.* 1.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., *Secretary*, Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole st., Cavendish sq. S. (Oct.) 1883-5. *Referee*, 1879-83. *Trans.* 2.

*Elected*

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde park. C. 1882-3. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., LL.D., *Vice-President*, Consulting Physician to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to the Middlesex Hospital, and to the Royal Hospital for Diseases of the Chest; 35, Bruton Street, Berkeley square.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital; 4, Sackville street, Piccadilly.

1877 PYE-SMITH, PHILIP HENRY, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Examiner in Physiology at the University of London; 54, Harley street, Cavendish square.

1850 †QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to University College Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Referee*, 1845-6, 1848, 1858-9. *Lib. Com.* 1846. *Trans.* 1. *Pro.* 2.

1852 †RADCLIFFE, CHARLES BLAND, M.D., *Treasurer*, Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879-80. T. 1881-5. *Referee*, 1862-6, 1870-8.

*Elected*

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. *Referee*, 1885.

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Referee*, 1860-72, 1875-81. *Trans.* 1.

1882 REID, JAMES, M.D., Resident Physician to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; 34, St. George's place, Canterbury.

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., *Vice-President*, Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1881 RICE, GEORGE, M.B.; C.M., Sutton, Surrey.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., 13, Nelson crescent, Ramsgate.

1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.

1863 RINGER, SYDNEY, M.D., F.R.S., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans.* 6.

*Elected*

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Surgery at, the London Hospital; 22, Finsbury square. C. 1885. *Trans.* 2.

1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 11, St. John street, Deansgate, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; 53, Harley street, Cavendish square.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1843 RODEN, WILLIAM, M.D., Morningside Lodge, Kidderminster.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road [19, Ovington gardens, S.W.]. C. 1879-80.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Assistant Surgeon to King's College Hospital; 50, Harley street, Cavendish square.

1882 ROUTH, AMAND J. McC., M.D., B.S., Physician to the Samaritan Free Hospital for Women; Assistant Physician Accoucheur to the Charing Cross Hospital; Obstetric Physician to the St. Marylebone General Dispensary; 6, Upper Montagu street, Montagu square.

1849 †ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

*Elected*

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.

1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1879 SANGSTER, ALFRED, B.A., M.B., Physician to the Skin Department, and Lecturer on Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.

1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Boreatton park, Baschurch, near Shrewsbury.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. *Trans.* 2.

1845 †SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1879 SAVAGE, GEORGE HENRY, M.D., Bethlem Royal Hospital, St. George's road, Southwark.

*Elected*

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 6.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street.

1873 SCOTT, JOHN MOORE JOHNSTON, M.D., Lurgan, County Armagh.

1861 *SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. C. 1884-5. *Trans.* 2.

1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 59, Welbeck street, Cavendish square. *Trans.* 1.

1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square. *Sci. Com.* 1879.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1882 SHARKEY, SEYMOUR J., M.B., Assistant Physician, Joint Lecturer on Pathology, and Demonstrator of Morbid Anatomy, to St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Referee*, 1842-3, 1846-50, 1855-7, 1865. *Lib. Com.* 1843. *Trans.* 4.

*Elected*

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., House Surgeon, St. George's Hospital.

1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1848 †SIEVEKING, EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1842 †SIMON, JOHN, C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1882 SMITH, CHARLES JOHN, 54, Old Steyne, Brighton.

1879 SMITH, E. NOBLE, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 5, George street, Hanover square.

1885 SMITH, JAMES GREIG, M.B., C.M., Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton.

1872 SMITH, T. GILBART, M.A., M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. *Trans.* 1.

1866 SMITH, HEYWOOD, M.A. M.D., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 18, Harley street, Cavendish square.

*Elected*

1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. *Referee*, 1873-4, 1880-5. *Sci. Com.* 1867. *Trans.* 3.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, M.D., F.R.S. Ed., Physician to the Dispensary, Cheltenham; Bayshill Villa, Cheltenham.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1875 SPITTA, EDMUND J., Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1882 STEAVENSON, WILLIAM EDWARD, M.D., 39, Welbeck street, Cavendish square.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board.

1884 STEWART, EDWARD, M.D., 16, Harley street.

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1879 *STIRLING, EDWARD CHARLES, late Assistant Surgeon and Lecturer on Physiology at St. George's Hospital; Adelaide, South Australia [care of T. Gemmell, Esq., 11, Essex street, Strand].

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, WILLIAM, M.D., Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

*Elected*

1884 STONHAM, CHARLES, Curator of the Anatomical Museum, University College, London, and Assistant Surgeon to the Cancer Hospital, Brompton; 109, Gower street.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Professor of Clinical Ophthalmic Surgery in University College, and Senior Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5. *Lib. Com.* 1867-8.

1871 STRONG, HENRY JOHN, M.D., Whitgift House, George street, Croydon.

1863 †STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. *Referee*, 1882-5.

1871 †SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1883 SUTTON, JOHN BLAND, Lecturer on Comparative Anatomy and Senior Demonstrator of Anatomy, Middlesex Hospital Medical College; 22, Gordon street, Gordon square. *Trans.* 2.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health, Oldham; Higher Broughton, Manchester.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 4.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

*Elected*

1875 TAY, WARREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Trans.* 1.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 9.

1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Referee*, 1876-7. *Trans.* 1.

1857 THOMPSON, HENRY, M.D., Consulting Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

1852 †THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 7.

1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1881 THOMSON, WILLIAM SINCLAIR, M.D., 40, Ladbroke grove, Kensington park gardens.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Trans.* 2.

*Elected*

1883 THURSFIELD, THOMAS WILLIAM, M.D., 26, The Parade, Leamington.

1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

1872 TOMES, CHARLES S., B.A., F.R.S., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square. *Lib. Com.* 1879.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1882 TOOTH, HOWARD HENRY, M.B., Assistant Demonstrator of Practical Physiology, St. Bartholomew's Hospital; 34, Harley street, Cavendish square.

1871 *TREND, THEOPHILUS W., M.D., Raeberry Lodge, Southampton.

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 18, Gordon square. *Trans.* 3.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the Royal Sea Bathing Infirmary for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, late Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the North-Eastern Hospital for Children, and to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., San Remo, Italy.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Joint Lecturer on Practical Surgery at St. George's Hospital; 49, Green street, Park lane.

*Elected*

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Physician for the Diseases of Women, West London Hospital; Physician to the Victoria Hospital for Children, Chelsea; 8, Upper Brook street, Grosvenor square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 19A, Hanover square.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

1884 WAKLEY, Thomas, Jun., 96, Redcliffe gardens.

1868 *WALKER, ROBERT, Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1883 WALLER, AUGUSTUS, M.D., 29, Abbey road, St. John's wood.

1867 *WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. *Lib. Com.* 1882-5. *Trans.* 3.

*Elected*

1852 †WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 41, Hyde park square. C. 1872. *Trans.* 1.

1883 *WALTERS, JAMES HOPKINS, 43, Castle street, Reading.

1851 †WALTON, HAYNES, Senior Surgeon to St. Mary's Hospital, 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, Bristol.

1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton gardens, Maida vale. *Referee*, 1881-5.

1877 WARNER, FRANCIS, M.D., Assistant Physician to the London Hospital and to the East London Hospital for Children; Lecturer on Botany at the London Hospital; 24, Harley street, Cavendish square. *Trans.* 1.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

1879 WATERS, JOHN HENRY, M.D., C.M., 101, Jermyn street.

1878 WATNEY, HERBERT, M.D., 1, Wilton crescent, Belgrave square, and Buckhold, Basildon, Reading.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., Medical Electrician to St. Mary's Hospital; 30, Welbeck street, Cavendish square.

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

*Elected.*

1840 WEBB, WILLIAM WOODHAM, M.D., 82, Avenue des Termes, Paris.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., *Vice-President*, Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1878 WEISS, HUBERT FOVEAUX, 11, Hanover square.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

1854 WELLS, SIR THOMAS SPENCER, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 13. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 55, Harley street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Physician to the Royal Free Hospital; Medical Registrar and Medical Tutor to St. Bartholomew's Hospital; 15, Wimpole street, Cavendish square. *Trans.* 3.

1882 WHARRY, CHARLES JOHN, M.D., Resident Superintendent, Government Civil Hospital, Hong Kong.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.

1828 WHATLEY, JOHN, M.D.

*Elected*

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 WHITE, JOHN.

1881 WHITE, WILLIAM HALE, M.D., Assistant Physician to Guy's Hospital; 4, St. Thomas's street, Southwark. *Trans.* 1.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Surgeon to the Manchester Royal Infirmary; Senior Surgeon to the Manchester and Salford Lock and Skin Hospital; 24, St. Ann's square, Manchester. *Trans.* 1.

1885 *WHITLA, WILLIAM, M.D., Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham park, Kent.

1883 *WILKINSON, THOMAS MARSHALL, Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 7, Lindum road, Lincoln.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Consulting Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

1883 *WILLANS, WILLIAM BLUNDELL, Great Hadham, Herts.

1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. *Referee*, 1882-5. *Trans.* 2.

*Elected*

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]; Villa de Rocher, Cannes. C. 1849-50. V.P. 1860-1. P. 1873-4. *Referee*, 1843-4. *Sci. Com.* 1862. *Trans.* 1.

1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 47, Upper Brook street, Grosvenor square. C. 1884-5. *Lib. Com.* 1880-3. *Trans.* 3.

1881 WILLIAMS, DAWSON, M.D., 4, Oxford and Cambridge Mansions, Marylebone road.

1872 WILLIAMS, JOHN, M.D., Obstetric Physician to University College Hospital; Examiner in Obstetric Medicine at the University of London; 11, Queen Anne street, Cavendish square. *Referee*, 1878-85. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 19, Whitehall place.

1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

1825 WISE, THOMAS ALEXANDER, M.D.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1885 WOLFENDEN, RICHARD NORRIS, M.D., 19, Upper Wimpole street.

*Elected*

1851 †WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; Examiner in Surgery in the University of London; 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-85. *Lib. Com.* 1866. *Trans.* 3.

1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., Rockhampton, Queensland.

1881 WOODMAN, SAMUEL, Consulting Surgeon to the Ramsgate and St. Lawrence Royal Dispensary; 5, Prospect terrace, Ramsgate.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1865 WOTTON, HENRY, M.D., 15, Notting Hill terrace, Kensington.

1878 YEO, GERALD F., M.D., M.C., Professor of Physiology in King's College, London; Examiner in Physiology, University of London; King's College, Strand.

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[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

## HONORARY FELLOWS.

(Limited to Twelve.)

*Elected*

1883 CARPENTER, WILLIAM BENJAMIN, C.B., M.D., LL.D., F.R.S., Corresponding Member of the Institute; 56, Regent's park road.

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Professor of Chemistry in the Royal School of Mines; Corresponding Member of the French Institute; Royal College of Chemistry, South Kensington Museum, and the Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., D.C.L., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Bromley, Kent.

*Elected*

1847 OWEN, SIR RICHARD, K.C.B., D.C.L., LL.D., F.R.S., late Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

1883 PARKER, WILLIAM KITCHEN, F.R.S., Hunterian Professor of Comparative Anatomy in the Royal College of Surgeons; Crowland, Trinity road, Upper Tooting.

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge; Secretary to the Royal Society, &c.; Lensfield Cottage, Cambridge.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

## FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

*Elected*

1878 BACCELLI, GUIDO, M.D., Professor of Medicine at Rome.

1883 BIGELOW, HENRY J., M.D., Professor of Surgery at Harvard University, and Surgeon to the Massachusetts General Hospital.

1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna; Vienna.

1883 CHARCOT, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Quai Malaquais 17, Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1883 DUBOIS REYMOND, EMIL, M.D., Professor in Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1873 HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics; Berlin.

1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.

1856 LANGENBECK, BERNHARD, M.D., late Professor of Surgery in the University of Berlin.

*Elected*

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France (Academy of Sciences).

1878 SCANZONI, FRIEDREICH WILHELM VON, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.

1856 VIRCHOW, RUDOLPH, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

# LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

## DATE OF ELECTION.

1833 Sir George Burrows, Bt., M.D., F.R.S.
Thomas A. Barker, M.D.
1835 Richard Quain, F.R.S.
Thomas A. Nelson, M.D.
1836 Alexander Shaw.
J. George French.
1837 Thomas Blizard Curling, F.R.S.
George Busk, F.R.S.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Chas. J. B. Williams, M.D., F.R.S.
Charles Hutton, M.D.
Samuel A. Lane.
Sir James Paget, Bt., F.R.S.
1841 Sir Henry A. Pitman, M.D.
Sir William Bowman, Bart., F.R.S.
John Parkin, M.D.
Paul Jackson.
1842 Charles West, M.D.
Frederic Weber, M.D.
John Simon, C.B., F.R.S.
John Erichsen, F.R.S.
Sir Oscar M. P. Clayton.
1843 Robert Greenhalgh, M.D.
Sir Prescott G. Hewett, Bt., F.R.S.
Henry Lee.
Wm. White Cooper.
Luther Holden.
Edward Newton.
1844 Arthur Farre, M.D., F.R.S.
William Wegg, M.D.
1844 Thomas King Chambers, M.D.
Edwin Humby.
1845 Samuel Cartwright.
George D. Pollock.
Thomas Taylor.
Sir Edwin Saunders.
William Oliver Chalk.
Edward U. Berry.
Benjamin Ridge, M.D.
1846 John A. Bostock.
Barnard Wight Holt.
Carsten Holthouse.
1847 W. H. O. Sankey, M.D.
George Johnson, M.D., F.R.S.
1848 Edward H Sieveking, M.D.
Edward Ballard, M.D.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
John Clarke, M.D.
John Gregory Forbes.
1849 Hugh J. Sanderson, M.D.
C. H. F. Routh, M.D.
Edmund L. Birkett, M.D.
George T. Fincham, M.D.
Sir William W. Gull, Bt., M.D., F.R.S.
1850 Richard Quain, M.D., F.R.S
George Roper, M.D.
1851 Sir Wm Jenner, Bt., M.D., F.R.S.
H. Haynes Walton.
John Birkett.
John A. Kingdon.
Peter Y. Gowlland.
John Marshall, F.R.S.

1851 John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
George Gaskoin.
1852 C. Bland Radcliffe, M.D.
Walter H. Walshe, M.D.
William Adams.
John Cooper Forster.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Alfred Baring Garrod, M.D., F.R.S.
Samuel O. Habershon, M.D.
Sir Thomas Spencer Wells, Bt.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
Walter John Bryant, M.D.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Henry Thompson, M.D.
Hermann Weber, M.D.
George Owen Rees, M.D., F.R.S.
John Whitaker Hulke, F.R.S.
John Morgan.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
William Chapman Begley, M.D.
John William Ogle, M.D.
Wilson Fox, M.D., F.R.S.
John Fremlyn Streatfeild.
1859 Wm. Howship Dickinson, M.D.
William Scovell Savory, F.R.S.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Sir Andrew Clark, Bt., M.D., F.R.S.
Sigismund Sutro, M.D.
William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
William Henry Holman.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
1862 Thomas H. Tuke, M.D.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
William Henry Brace, M.D.
George Cowell.
Robert Farquharson, M.D., M.P.
M. Berkeley Hill.
1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D., F.R.S.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley M.D.
Walter John Coulson.
Thomas William Nunn.
Francis Mason.
Jos. Gillman Barratt, M.D.
1865 Charles Robert Drysdale, M.D.
James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.
George Fielding Blandford, M.D.
Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
Henry Wotton.
1866 Thomas Fitzpatrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
John Crockett Fish, M.D.
William Selby Church, M.D.
Edward John Waring, M.D.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
John Astley Bloxam.

1867 Charles Arthur Aikin.
Samuel Hill, M.D.
1868 H. Charlton Bastian, M.D., F.R.S.
William Henry Broadbent, M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
John Cockle, M.D.
Sir Thos. Crawford, K.C.B., M.D.
T. Henry Green, M.D.
William Rhys Williams, M.D.
Walter Moxon, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
William Henry Freeman.
1869 Joseph Frank Payne, M.D.
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P. Henry Pye-Smith, M.D.
Edward Nettleship.
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William T. Whitmore.
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Clinton T. Dent.
John H. Morgan.
Walter Pye.
Gerald F. Yeo, M.D.
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Hubert Foveaux Weiss.
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1879 Alfred Sangster, M.B.
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Edmund Owen.
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Horatio Donkin, M.B.
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William Watson Cheyne.
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George Henry Makins.
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1881 Cecil Yates Biss, M.D.
Richard Clement Lucas.
Stephen Mackenzie, M.D.
James Anderson, M.D..
William Hale White, M.D.
Eustace Smith, M.D.
William Sinclair Thomson, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.A.
Audley Cecil Buller.
W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.A., M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
C. B. Lockwood.
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John Barclay Scriven.
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Howard Henry Tooth, M.B.
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Joseph Mills.
A. T. Myers, M.D.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
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William Lang.
Henry Radcliffe Crocker, M.D.
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D. Astley Gresswell, M.B.
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Walter Hamilton Aciand Jacobson.
Edward Joshua Edwardes, M.D.
Walter H. H. Jessop, M.B.
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Victor A. Horsley.
Dudley Wilmot Buxton, M.D.
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Henry Roxburgh Fuller, M.B.
Wilmot Parker Herringham, M.B.
Augustus Waller, M.D.
William Pasteur, M.D.
Edward Albert Schäfer, F.R.S.
John Bland Sutton.
William Rose, M.B.

1883 Storer Bennett.
Henry Maudsley, M.B.
Robert Marcus Gunn, M.B.
James Dixon Bradshaw, M.B.
1884 George Newton Pitt, M.D.
Charles Stonham.
Stanley Boyd, M.B.
William Arbuthnot Lane, M.S.
Dennis Dallaway.
Thomas Whitehead Reid.
Arthur Marmaduke Sheild, M.B.
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Sidney Harris Cox Martin, M.B.
Wayland Charles Chaffey, M.B.
George Lawson.
Heneage Gibbes, M.D.
Thomas Wakley, Jun.
Robert James Lee, M.D.
F. Swinford Edwards.
Herbert Tyrrell Griffiths, M.D.
James Johnston, M.D.
Arthur Oakes, M.D.
Edward Stewart, M.D.
William A. Duncan, M.D.

1884 Charles Chinner Fuller.
Lovell Drage.
Jean Samuel Keser, M.D.
Charles Egerton Jennings, M.S.
George Richard Turner Phillips.
Bilton Pollard.
1885 Alexander Haig, M.B.
Wm. Dobinson Halliburton, M.D.
Theodore Dyke Acland, M.D.
Kenneth William Millican.
Frederick Walker Mott, M.B.
William Maunsell Collins, M.D.
James Berry.
John Cahill.
Francis Henry Hawkins, M.B.
John Poland.
James Greig Smith.
John Mackern, M.D.
George Gulliver, M.B.
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Edward Emanuel Klein, M.D., F.R.S.
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## ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

## Regulations relative to the publication of the 'Proceedings of the Society.'

That, as a general rule, the 'Proceedings' will be issued every two months, subject to variations dependent on the extent of matter to be printed.

That a Copy of the 'Proceedings' will be sent, postage free, to every Fellow of the Society resident in the United Kingdom.

That the 'Proceedings of the Society' may be obtained by non-members at the Society's House, 53, Berners Street, on pre-payment of an annual subscription of five shillings, which may be transmitted either by post-office order or in postage-stamps; —this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent, carriage free, through a bookseller, or by post, the receiver paying the foreign charges.

That a notice of every paper will appear in the 'Proceedings.' Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the 'Proceedings' *only*, or in the 'Proceedings' and 'Transactions;' and in all cases they will be expected to furnish an Abstract of the communication.

The Abstracts of the papers read will be furnished to the Journals as heretofore.

# ADDRESS

OF

## GEORGE JOHNSON, M.D., F.R.S.,

PRESIDENT,

AT THE

## ANNUAL MEETING, MARCH 2ND, 1885.

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GENTLEMEN,—If I were to estimate the experience of former Presidents of the Society by a comparison with my own, I should conclude that they must have found the preparation for the annual meeting the most arduous and anxious work of the official year. For surely the annual address, which, in accordance with custom and precedent, is mainly composed of obituary notices of recently deceased Fellows, cannot have been found an easy task by any, even the ablest and most eloquent, of the many eminent men who have preceded me in this chair.

One of the most distinguished of our foreign Honorary Fellows, the late Dr. Samuel D. Gross, of Philadelphia, in the course of an eloquent and affectionate memoir of his friend and fellow-countryman, Dr. Valentine Mott, refers to the difficulties which beset the path of a contemporary biographer in the following terms:—"It is confessedly difficult under any circumstances to write the biography of a contemporary. For, on the one hand, there is great danger of indulgence in fulsome eulogy, and, on the

other, of being blinded by jealousy and prejudice. In either event injustice is apt to be done alike to the subject and to the truth of history." Whether I have succeeded in my endeavour to avoid these two opposite but equally faulty extremes I must leave to the judgment of others.

As on previous occasions it has been a source of satisfaction to me to observe that obituary notices of intimate friends which I have myself contributed to one or other of the medical journals have been utilised by former Presidents of the Society, so now, in addition to such personal knowledge as I possess of our deceased Fellows and their published writings, I have to acknowledge my indebtedness to the obituaries which have appeared in the public journals. In some instances, too, I have been much assisted by information which has been kindly afforded me by relatives or intimate friends of the deceased.

Since our last annual meeting the names of fourteen of our Fellows have been erased from our muster-roll by the hand of death. Of these six were *resident* Fellows, namely, Dr. Barclay, Dr. Fairlie Clarke, Mr. Cæsar Hawkins, Sir Erasmus Wilson, Dr. David Alexander King, and Dr. Baxter. Six were *non-resident* Fellows, namely, Dr. Samuel Best Denton, Dr. Lashmar, Mr. John Arnold, Mr. James Stock Daniel, Dr. William Gillett Cory, and Mr. William Collins Worthington. One *Honorary* Fellow, Dr. Allen Thomson, and one *Foreign Honorary* Fellow, Dr. Samuel D. Gross, complete the list of our losses.

It may be stated in general terms that, while most of those whose deaths we have to deplore had attained to a good old age and, having accomplished their life's work, had retired from the active duties of their profession, in at least three instances men whose past performances had led to a well-founded hope of an honorable and brilliant future, have been cut off in mid-career and at a comparatively early age. I refer particularly to Dr. Fairlie Clarke, Dr. Baxter, and Dr. King.

I propose now to speak of each of our deceased Fellows in the order in which their deaths occurred.

*Dr. Samuel Best Denton* was born on the 3rd May, 1797, and having received his early education at Hull he entered at the then united Guy's and St. Thomas's Hospitals. He became a Licentiate of the Society of Apothecaries in 1820, a Member of the College of Surgeons in 1843, and M.D. of Aberdeen in 1852. He was elected a Fellow of this Society in 1846. After practising at Hornsey, near Hull, for upwards of sixty years, he died March 9th, 1884, having nearly completed his eighty-sixth year.

His partner, Mr. James Thomas Jones, in a private note with which I have been favoured, states that from his youth Dr. Denton was an ardent student not only of subjects strictly professional, but also of astronomy, botany, and other branches of natural history. He had the gift of forming a rapid and generally a correct diagnosis, and in his treatment of disease he displayed much care, skill, and foresight. He was an admirable specimen of the old English gentleman, never displaying this character to greater advantage than when in the homes of the poor. He was a man of the purest truth and integrity, and his memory is deeply loved and revered. A local paper says of him:—"His old friend, Sir James Alderson, often advised him to migrate to the metropolis as a more suitable field for his talents, but being of a quiet, retiring nature he preferred to remain in this somewhat secluded locality. Every respect was shown by the townspeople during the time of his funeral, all the shops being closed and business suspended, while the streets and the church were thronged with sorrowing patients and friends."

Dr. Denton published in the 'Provincial Medical Journal' a paper on "Hydrophobia and Chloroform," and in the 'Lancet' one on "Enlargement of the Prostate Gland," and another on "Tubercular Disease of the Periosteum."

*Dr. Allen Thomson*,[1] who was elected an Honorary Fellow in 1883, was born in Edinburgh on the 2nd April,

[1] For the main facts of Dr. Allen Thomson's career I am indebted to an interesting memoir by Dr. McKendrick, read before the Philosophical Society of Glasgow, April 30th, 1884, and to an obituary in the 'Lancet,' April 12th, 1884.

1809. He was educated at the High School and the University of his native city. As a student his merits were recognised, as those of his father had been before him, in his election to the Presidency of the Royal Medical Society. In 1830 he took the degree of Doctor of Medicine in the University of Edinburgh, when the bent of his mind towards embryology was shown by his Graduation Thesis, "On the Development of the Heart and Blood-vessels in Vertebrate Animals." After taking his degree he went for a fifteen months' course of study to Paris, where he attended the various hospitals, and amongst other lectures those of Cuvier.

In 1831 he obtained the Fellowship of the Royal College of Surgeons of Edinburgh, and became associated with the late Dr. William Sharpey in a course of systematic lectures on Anatomy and Physiology in the Extra-Academical School. This association continued from 1831 to 1836, when Dr. Sharpey was appointed Professor of Physiology in University College, London. The next three years, from 1836 to 1839, his health having somewhat failed from overwork, Dr. Allen Thomson spent on the Continent with the noble family of Bedford.

In 1839 he was appointed Professor of Anatomy in the Marischal College and University of Aberdeen, an appointment which he resigned in 1841 to become again a teacher of anatomy in the Extramural School in Edinburgh. In that year Professor Alison resigned the Chair of Institutes of Medicine (Physiology) in the University of Edinburgh and in 1842 Dr. Allen Thomson was appointed his successor. He held the Edinburgh professorship for six years, when he was appointed by the Crown in 1848 to succeed Dr. James Jeffray in the Chair of Anatomy in the University of Glasgow, a professorship which he held with great distinction for twenty-nine years, resigning it in 1877, when he was succeeded by its present distinguished occupant, Dr Cleland, who in former years had been one of his demonstrators. During these thirty-five years he had the unique experience of being a professor

in three out of the four Scotch universities, and in all of them he worked with an indefatigable industry, not merely in connection with the immediate duties of his chair, but as a contributor to scientific literature, and at the end of his days he had become generally known throughout the scientific world as one of the most careful, judicious, accurate, and learned investigators and teachers of his favourite subjects.

It was in the field of embryology that he won his laurels, and few if any men have done so much to render this department of biological science familiar to British naturalists. He wrote largely for the 'Cyclopædia of Anatomy and Physiology,' edited by the late Dr. Todd. The articles on "Circulation," "Generation," and "Ovum," are his, and to the past and current editions of the 'Encyclopædia Britannica' he contributed the articles on these and other kindred subjects. He also wrote on physiological optics, more especially on the mechanism by which accommodation for vision at different distances is effected. His name has long been associated with Quain's 'System of Human Anatomy,' as editor especially of the descriptive parts of the seventh and eighth editions. In the seventh edition he was associated with Professors Sharpey and Cleland, in the eighth with Professors Sharpey and Schäfer, and in the ninth and last edition with Professors Schäfer and Thane, of University College. He brought out a second edition of his father's 'Life of Cullen.' To the Royal Societies of London and Edinburgh, and to British and Foreign journals, he contributed numerous special papers and articles. To the Royal Society alone he contributed about twenty papers.

During his distinguished career he received many scientific honours. He was elected a Fellow of the Royal Society of Edinburgh in 1838, and of the Royal Society of London in 1848. For eighteen years, from 1859 to 1877, he was a Member of the Medical Council for the Universities of Glasgow and St. Andrew's jointly. In 1872 he was President of the Biological Section of the

British Association at the meeting in Edinburgh, and in 1876, he had the honour of being elected President of the Association. At the meeting at Plymouth, in 1877, his address on his favourite topic, "The Development of the Forms of Natural Life," was a masterly history of the gradual acceptance of the doctrines connected with the name of Darwin, whose important generalisations his open and receptive mind had long before accepted.

In 1871 he received from the University of Edinburgh the degree of LL.D., and in 1877 the Glasgow University conferred on him the same degree. In 1882 he received the degree of D.C.L. from the University of Oxford.

As a citizen of Glasgow Dr. Allen Thomson took a prominent part in various great public undertakings, especially as Chairman of the Removal and Buildings Committee of the University of Glasgow from 1863 to 1874, which led to the erection of the grand university buildings on Gilmore Hill, and the carrying out of this great scheme is said to have been largely due to his energy and tact. He also took an active part in the erection of the Western Infirmary of Glasgow. This noble institution is a model hospital, and Dr. Allen Thomson, as a Member of the Board of the Directors, did much to make it what it is.

On his retirement from the University of Glasgow in 1877 his portrait, painted by the President of the Scottish Academy of Arts, the late Sir Daniel Macnee, was presented by his friends and admirers to the University, and it now hangs in the Hunterian Museum of that University.

He was appointed a Trustee of the Hunterian Museum of the Royal College of Surgeons in succession to the late Dr. Sharpey.

Dr. Thomson was in his usual good health until within about four months of his death. About the middle of December, 1883, he began to complain of his left eye, which was found to have become glaucomatous, and on December 15th Mr. John Couper, with the concurrence of Sir Wm.

Bowman, performed iridectomy. The operation was successful in relieving pain, restoring normal tension, and maintaining vision. About a fortnight later he began to suffer from lancinating pains, as he said, in the course of the left temporo-auricular nerve. During the last few weeks of his life I had the melancholy privilege of attending him with Mr. Couper and Mr. Aikin. The next serious symptom was sudden loss of vision in the right eye. This was seen to be associated with plugging of the main branch of the arteria centralis retinæ. As there was no evidence of cardiac valvular disease it is probable that the blood coagulation had occurred in a vessel altered by senile degeneration of its walls, and the neurotic symptoms which followed were probably due to a similar obstruction in other vessels. There was a temporary and partial paralysis of the left hand; then the muscles on the right side of the face were affected; next the vagus became implicated, and there followed almost incessant hiccough, dysphagia, and lastly dyspnœa, which proved fatal on March 22nd, 1884.

The remains, which were taken to Scotland for burial, were followed by a number of distinguished scientific friends to Euston Station, where they were met by many others who had assembled to show their respect and esteem for the deceased.

Dr. Allen Thomson will long be held in affectionate remembrance not only for the extent and variety of his scientific attainments, but for his wisdom in council, the genuine kindly courtesy which gave an indescribable charm to his manner, and the enduring warmth of his friendship.

*Dr. Charles Lashmar*, of Croydon, was elected a Fellow of this Society in 1841, his proposal paper having been signed by Thomas Addison, Richard Bright, and Bransby Cooper. He obtained the license of the Apothecaries' Company in 1827, and the M.D. of Erlangen in 1841. Having practised at Croydon for more than forty-five years he retired some years since to Brighton, where he

died on March 25th, 1884, at the age of eighty. I am not aware that Dr. Lashmar made any contribution to the literature of the profession.

*Dr. Andrew Whyte Barclay*[1] was descended from an old Scotch family, his father having been a naval officer during the earlier years of the present century. Born at Desart in Fifeshire in 1817, he received his preliminary education at the Edinburgh High School, commenced the study of medicine in the University of Edinburgh in 1834, and graduated M.D. in 1839. He afterwards studied in Berlin, and subsequently, with two of his brothers, visited Italy and France. He then went to Cambridge and entered Caius College, where he obtained several scholarships.

In 1847 he graduated M.B. at Cambridge, and soon after placed his name on the books of St. George's Hospital, where he was appointed Medical Registrar. His reports of cases during his tenure of that office are said to be of great value. In 1851 he was elected a Fellow of the Royal College of Physicians, and in the following year he graduated M.D. at Cambridge. In 1857 he was appointed Assistant Physician to St. George's, and in 1862 he succeeded Dr. Bence Jones as full physician, which office he resigned in 1882 in accordance with the laws of the hospital.

Dr. Barclay held in succession two important lectureships,—that on Materia Medica and that on the Principles and Practice of Physic. As Physician to the hospital and as a teacher his work was conscientiously and faithfully performed. As Treasurer to the Medical School during a number of years he was brought into close contact with the pupils and was by them much esteemed.

At the College of Physicians, where his business habits were highly appreciated, he did good work as Councillor, Examiner, and Censor, and a few months before his death he was chosen for the important office of Treasurer of the College. In 1881 he gave the Harveian Oration, and I

[1] 'Lancet,' May 10th, 1884, and 'Medical Times and Gaz.,' May 17th, 1884.

scarcely need remind you that in the same year he was elected President of this Society.

Dr. Barclay was the author of several works: 'The Progress of Sanitary Measures and Preventive Medicine,' 'A Manual of Medical Diagnosis,' a third edition of which was published in 1870; 'Gout and Rheumatism in Relation to Disease of the Heart.' His small volume on 'Medical Errors' involved him in some controversy, since not all of his contemporaries to whom he imputed error admitted the justice of the imputation. Our 'Transactions' contain two valuable papers on "Statistics of Valvular Disease of the Heart" (vols. xxxi and xxxv); and the articles on "Delirium Tremens" and on "Croup and Diphtheria" in 'Holmes's System of Surgery,' 1860, were from Dr. Barclay's pen. For a number of years Dr. Barclay was Medical Officer of Health for the parish of Chelsea, and during the latter part of his life his attention was chiefly given to sanitary subjects. His death occurred after so short an illness as to give a painful shock to his numerous friends. I saw him apparently in his usual good health at a meeting of the College of Physicians on Thursday the 24th April; he was taken ill that evening and died on the morning of the 28th, the immediate cause of death being cardiac failure consequent on intestinal irritation.

Dr. Barclay was highly esteemed for his admirable social qualities, and it has been said of him that "those who knew him best loved him most." He is also deserving of respect for the courage with which he would contend for a doctrine which he knew to be unpopular but which he conscientiously believed to be true. In illustration of this I may refer to an elaborate paper of his published thirteen years ago ('Lancet,' March 2nd, 1872), in which he contends, in opposition to the high authorities whom he quotes, that the so-called pre-systolic or, as Dr. Gairdner terms it, the auricular-systolic murmur at the apex of the heart is not a result of the blood passing into the ventricle through a constricted or roughened mitral orifice, but that it

is a regurgitant murmur the result of mitral incompetence, and that it is systolic in rhythm. In this contention I believe that Dr. Barclay was in error, but he nevertheless deserves credit for having the courage of his opinion.[1]

In contrast with the courage thus displayed by Dr. Barclay, I venture to remark in passing that there are two forms or phases of intellectual cowardice whose tendency is to retard the progress of medical as of other kinds of knowledge. The one is a dread of, and therefore a shrinking from, the advocacy of a doctrine believed to be true, but which is not in accordance with the opinion of the majority; the other is an unwillingness to investigate facts and to weigh arguments, the tendency of which might be to prove that doctrines to which we have publicly professed our assent, may prove to be erroneous, and therefore demand from us an equally public acknowledgment of our error. As an example of one who to the very end of his prolonged life displayed in a pre-eminent degree the moral courage which is the direct opposite of the cowardice here alluded to, I need only mention the revered name of the late Sir Thomas Watson.

*Mr. John Arnold* was elected a Fellow of this Society in 1878. He was the son of a planter in British Guiana. He was educated at Stonyhurst College, and matriculated at the University of London in 1863. He was originally intended for the army, but having failed to pass the entrance examination at Woolwich, he entered and went through the usual course at St. Bartholomew's.

[1] That a diastolic murmur, accompanied by a thrill leading up to and terminating in the impulse of the ventricle, is a result of mitral constriction or roughening of the auricular surface of the valves, was clearly pointed out by Skoda ('On Auscultation and Percussion,' Dr. Markham's translation, p. 232), and subsequently by Dr. Markham ('Diseases of the Heart,' 1st ed., p. 206, 2nd ed., p. 122), who quotes in support of the doctrine Hamernjk, Skoda and Jacksch. Dr. Markham speaks of the murmur as of "comparatively rare occurrence." Afterwards Dr. Gairdner suggested that since a murmur which immediately precedes the systole is in fact synchronous with the systole of the auricle it might well be designated auricular-systolic. Dr. Gairdner also showed that the murmur is much more common than previous writers had supposed.

Having become a Member of the College of Surgeons and a Licentiate of the Society of Apothecaries, he was for a year Obstetric Assistant under Dr. Greenhalgh. He then returned to Demerara and became Resident Surgeon of the Colonial Hospital there. During an epidemic of yellow fever he made some hundreds of post-mortem examinations, but I am not aware that the results have been published. During his tenure of office Dr. Craig, Surgeon-General of Trinidad, visited the Demerara Hospital and was so favorably impressed by Mr. Arnold that he shortly afterwards offered him the post of Medical Officer of Health for Trinidad and Surgeon to the Colonial Hospital there. He discharged the duties of these offices to the entire satisfaction of the Surgeon-General and other official superiors, and obtained also a large private practice.

He was more than once compelled to return to Europe on account of his health, and he died last summer of tubercular disease while on a visit to Aix-les-Bains. He is said to have been thoroughly well grounded in his profession and to have been respected and honoured by all who knew him.

*Dr. Samuel D. Gross*, who was elected a Foreign Honorary Fellow of this Society in 1868, was born near Easton in Pennsylvania, July 8th, 1805. After receiving a classical education he began the study of medicine at the age of nineteen, and having graduated M.D. in the Jefferson Medical College, Philadelphia, in 1828, he at once commenced the practice of his profession in that city. After the lapse of eighteen months, having meanwhile married, Dr. Gross returned to his native place, Easton, where he soon obtained a good practice, and where, to increase his knowledge of practical anatomy, he built a dissecting-room at the end of his garden and dissected daily for several hours. In October, 1833, he removed to Cincinnati, having accepted the office of Demonstrator of Anatomy in the Medical College of Ohio. After teaching anatomy for two sessions in the Medical College he was in 1835 unanimously elected to the Chair of Pathological

Anatomy in the Medical Department of the Cincinnati College. During the next few years he devoted most of his time to the study of Pathological Anatomy, the collecting and preservation of specimens, and laying the foundation of a museum of Morbid Anatomy. He thus acquired the knowledge which enabled him in 1839 to publish his 'Elements of Pathological Anatomy,' of which a second edition appeared in 1845, and the third and last in 1857.

In 1840 he accepted the Professorship of Surgery in Louisville Medical Institute, afterwards the University of Louisville. His class, which during the first session numbered 204, afterwards increased to 406. In 1843 he published a monograph 'On the Nature and Treatment of Wounds of the Intestines,' the result mainly of numerous experiments which he had performed on dogs. And he defends himself and others who have practised vivisection against the unwarrantable charge of cruelty, the result as he says of "the mawkish sentimentality of the Society for the Prevention of Cruelty to Animals which have made so much ado about this matter." In 1849 there was a dispute between the University and the City as to the government of the University, and at this critical period Dr. Gross, being in doubt as to the result of the suit, accepted the offer of the Chair of Surgery in the University of the city of New York rendered vacant by the resignation of Dr. Valentine Mott. He passed the winter of 1850-51 in the city of New York, but before the termination of the session he was solicited by his former colleagues at Louisville to resume his Chair in that University, the suit in the meantime having been decided in favour of the University. For various reasons, amongst others the earnest wish of his family to return to their former home, he was led to tender his resignation to the University of New York and to resume his Chair in that of Louisville.

In 1851 he published 'A Practical Treatise on the Diseases, Injuries, and Malformations of the Urinary Bladder, the Prostate Gland, and the Urethra,' and in 1854 his 'Practical Treatise on Foreign Bodies in the Air

Passages' was published. This work is so complete that if it were now republished it would require no other changes or additions than such as would be called for by the results of laryngoscopic work since the publication of the original treatise.

In 1856 Dr. Gross accepted the Chair of Surgery in his alma mater, the Jefferson Medical College of Philadelphia. A strong motive for taking this step was his desire to be relieved from a large family practice which left him little time for writing his elaborate 'System of Surgery,' upon which he had been engaged for several years.

In the autumn of 1859 the work was published in two large octavo volumes, and the sixth and last edition appeared in 1882.

During the war of the Rebellion, he took great interest in gunshot wounds, and visited many battle-fields to observe their peculiar features, of which he made valuable notes. In March, 1882, he resigned the Chair of Surgery which he had filled for twenty-six years, and he was thereupon unanimously elected Emeritus Professor.

In addition to the various works which have here been mentioned, Dr. Gross published innumerable papers, addresses, and memoirs in the various medical journals. He accomplished this vast amount of writing by rising early and working in an orderly and systematic manner. He said of himself that "his genius was the genius of industry, perseverance, and common sense," and the result has been that no previous medical teacher or author on the continent of America exercised so widespread and commanding an influence as Professor Gross. He was a member of numerous medical and scientific societies both American and foreign.

In 1872 the University of Oxford conferred on him the degree of D.C.L., in 1880 the University of Cambridge that of LL.D., and last year the University of Edinburgh conferred, *in absentiâ*, the degree of LL.D. In 1881 he was invited by Mr., now Sir William, Maccormac to deliver the address on behalf of the American delegates

to the International Medical Congress, but to his great regret he was unable to be present.

With the exception of slight rheumatic pains Dr. Gross had good health until a few months before his death, when he began to suffer from indigestion, swollen feet, and other symptoms of a weak heart. In January, 1884, he had a bronchial attack which confined him to the house. Being thus deprived of fresh air and exercise his appetite and digestive powers failed and he died from exhaustion on the 6th May last.

In accordance with his expressed wishes, the following day, after a funeral service in the presence of his family and attendants, his body was taken to the Crematory at Washington, Pa., and there reduced to ashes, which on the following Sunday were deposited in the family vault beside the coffin of his wife at Woodlands Cemetery.[1]

*Dr. William Fairlie Clarke*[2] was the son of an officer in the Civil Service of the Honorable East India Company, and he was born at Calcutta in 1833.

He was educated first at the High School of Edinburgh, then at Rugby under Dr. Goulburn, whence he proceeded to Christ Church, Oxford. After taking his B.A. degree he returned to Edinburgh with the intention of studying for the Bar, but finding medicine more to his taste he gave up the law, and in 1858 entered as a medical student at King's College. There he soon attracted the notice and acquired the esteem of his fellow-students and his teachers, who recognised in him a highly cultured and refined gentleman with deep and earnest religious convictions. After graduating M.A. and M.B. at Oxford in 1862 he returned to King's College, and for six months was House Surgeon of the Hospital, after which, for one year, he held the office of Assistant Demonstrator of Anatomy.

[1] For further particulars of Dr. Gross's distinguished career see an interesting memoir by Dr. I. Minis Hays, in the 'American Journal of the Medical Sciences' (Jan., 1884), and in the 'Transactions of the College of Physicians of Philadelphia' (third series, vol. vii).

[2] 'Lancet,' and 'Brit. Med. Journal,' May 17th, 1884.

In 1863 he obtained the Fellowship of the College of Surgeons. He then, for a time, travelled on the Continent with Lord Shaftesbury, and finally commenced practice as a pure Surgeon in Curzon Street. He became Clinical Assistant to Mr. (now Sir William) Bowman at Moorfields, an office which he held for three years. He also became in succession Surgeon to the St. George's and St. James's Dispensary; Assistant Surgeon to the West London Hospital, and finally in 1871 Assistant Surgeon to Charing Cross Hospital. In 1865 he published a 'Manual of the Practice of Surgery' which went through three editions. In 1866 he was elected a Fellow of this Society, and in our 'Transactions' for 1872 there is a paper of his on "A Case of Unilateral Atrophy of the Tongue," and in the volume for 1874 another paper on "Cases of so-called Ichthyosis Linguæ."

I am informed by an eminent Ophthalmic Surgeon that Fairlie Clarke's account of transverse calcareous opacity of the cornea ("On Some Rare Forms of Opacity of the Cornea," 'Brit. Med. Journ.,' Oct. 8th, 1870) is both original and valuable, and evinces careful and discriminative clinical observation. He continued to take special interest in diseases of the tongue, and he published a monograph on that subject in 1873. He also wrote the article on "Diseases of the Tongue" in Dr. Quain's 'Dictionary of Medicine.'

He was much interested in various philanthropic subjects. When quite a young man he recognised the great importance of improving the dwellings of the poor, and in conjunction with his friend Mr. Bosanquet he established one of the earliest associations for effecting this upon a sound financial basis. He also thought much and wrote well on hospital out-patient reform and provident dispensaries, on poor-law relief, on medical missions, and on the temperance question. Articles from his pen on these and kindred subjects appeared in the 'Edinburgh,' 'Quarterly,' and 'Fortnightly' Reviews and in 'Macmillan's Magazine.'

In 1870 he married a lady eminently fitted to be his

helpmate, and soon afterwards he removed to a larger house in Mansfield Street, where for some years he lived very happily. In time, however, he found that his income from private practice as a pure Surgeon did not keep pace with the requirements of an increasing family, and in 1876, having taken his M.D. at Oxford, he determined to leave London and engage in general practice at Southborough in Kent. There he spent the last eight years of his life beloved and respected by all who knew him, and, in addition to the faithful discharge of his strictly professional work, taking an active interest in local and general philanthropic movements, which he was ever ready to assist by his voice and his pen. In 1881 the village of Southborough, notwithstanding its great natural advantages, was not free from grave sanitary defects and Dr. Clarke was long prostrated by a severe attack of typhoid fever. This serious illness appears to have left some permanent mischief, and about two months before his death there arose symptoms of obscure brain disease which, making rapid progress, proved fatal, to the grief of his numerous friends, on the 8th May, 1884. His premature death excited much sympathy for his bereaved widow and his four sons.

*Mr. Cæsar Henry Hawkins* was born September 19th, 1798, at Bisley, in Gloucestershire. His father the Rev. Edward Hawkins, was Vicar of Bisley and the youngest son of Sir Cæsar Hawkins, who was for many years Surgeon to St. George's Hospital and Sergeant Surgeon to George II and George III. At the age of nine Mr. Cæsar Hawkins entered Christ's Hospital, where he remained six years. In 1814, soon after leaving the Blue-coat School, he was articled for five years to Mr. Sheppard, a practitioner at Hampton Court. In 1819 he became a pupil at St. George's Hospital when Sir Everard Home was the most eminent Surgeon and Mr. Brodie was acting as his assistant.

He obtained the diploma of the College of Surgeons in 1821, and the same year he was House Surgeon at the

Lock Hospital. The following year he was House Surgeon at St. George's. After this for some years he taught Anatomy in the Hunterian School in Great Windmill Street. He was appointed Surgeon to St. George's in 1829. From 1832 to 1834 he lectured with Dr. Seymour at St. George's on Medical Jurisprudence, and after this he lectured on Surgery, first with Mr. George Babington and then with Mr. Tatum, until 1874, after which he occasionally gave clinical lectures. On resigning the office of Surgeon to the Hospital in 1861 he was made Consulting Surgeon, and he was requested by his old pupils to sit for his bust, which was presented to his wife.

In 1874 Mr. Hawkins printed for private circulation two volumes entitled 'Contributions to Pathology and Surgery.' These consist of a collection of miscellaneous writings for the most part scattered through the transactions of medical societies and medical periodicals; they also contain some admirable clinical lectures. These two volumes afford conclusive evidence of his industry and learning, of his skill and eminence as a Surgeon, and of his success and influence as a clinical teacher.

Amongst other papers of great interest is a reprint of a paper in the thirty-fifth volume of our 'Transactions' "On a Successful Case of Colotomy," to which is appended a tabular statement of forty-four cases in which the operation had been performed by other Surgeons. This publication is believed to have contributed greatly to encourage resort to the operation, especially in cases of stricture of the sigmoid flexure of the colon. The paper affords some striking and instructive illustrations of the difficulty which attends the diagnosis of the exact seat and cause of intestinal obstruction.

One of the most interesting clinical lectures is that (in vol. i, p. 136) "On the First Successful Case of Ovariotomy in a London Hospital." This case occurred in the year 1846, when anæsthetics were unknown, and when there was no suspicion that the contact of atmospheric air

with the peritoneum could be injurious otherwise than by being of too low a temperature.

Mr. Hawkins's distinguished career is so well known that in this room it is scarcely necessary to say that the former Bluecoat boy and the apothecary's apprentice lived to obtain all the honours which his professional brethren could bestow upon a Surgeon of the greatest eminence. He was twice President of the Royal College of Surgeons, first in 1852 and again in 1861; for seventeen years he was an Examiner at the College. For some years he represented the College on the General Medical Council, and until his death he was a Trustee of the Hunterian Museum. In 1849 he delivered the Hunterian Oration, when H.R.H. the late Prince Consort honoured the College by his presence. This learned oration occupies the first place in the volumes of collected writings before referred to. For the usual period of five years he was Examiner in Surgery at the University of London.

On the death of Sir Benjamin Brodie he was appointed Sergeant Surgeon to the Queen, being the fourth of his family who had obtained the same distinction. And it is a noteworthy fact that he was consulted by four generations of the Royal Family.

He joined this Society in 1828, and contributed twelve important papers to the 'Transactions.' He served in succession the offices of Councillor, Referee, Vice-President, Treasurer, and in 1855-6 that of President. In 1852 he was President of the Pathological Society, and he had been a Fellow of the Royal Society since 1856.

Referring to some personal characteristics, the writer of an interesting and appreciative memoir in one of the journals ('British Medical Journal,' Aug. 16th, 1884) says of him, "His manners had little enough in common with the fashionable host, who 'with his arms outstretched as if he would fly grasps the new comer.' To tell the truth, many people complained of him as cold and stiff on a slight acquaintance, but on a closer intimacy all this vanished

and his genuine kindness of heart, his sincerity, and his trustworthiness endeared him to a large circle of friends."

Two of Mr. Cæsar Hawkins's elder brothers were the Rev. Edward Hawkins, D.D., well known as the Provost of Oriel from 1828 to 1882, and the late Dr. Francis Hawkins, Physician to the Middlesex Hospital, and Registrar in succession of the Royal College of Physicians and of the General Medical Council. It is not without interest to observe that of the famous Provost, and his apparent coldness and reserve, his biographer, in a recent number of the 'Quarterly Review' (October, 1883), speaks in terms almost identical with those which I have just now quoted as applied to the great Surgeon. The writer says of the Provost, "A constitutional dread of overstepping by a hair's-breadth the strict line of truth (so at least it seemed) not only guarded him effectually from anything approaching to sentimental outburst, but even kept in check ordinary expressions of warmth, restrained him—even unpleasantly if the truth must be told—while in converse with those whom he did love and trust, as if through fear of possibly overstating his feelings."

Then follow statements to show that beneath an exterior apparently cold and reserved "he had the warmest as well as the most feeling heart."

It will be evident from these extracts that between the mental and moral characteristics of the eminent Surgeon and the famous Oxford Provost there was a close fraternal resemblance. The brothers, who were deeply attached to each other, were not long separated by death. The Provost died on November 18th, 1882, having nearly completed his ninety-fourth year, and the Surgeon followed on July 20th, 1884, at the age of eighty-six. His death appears to have been an indirect result of an accidental bruise of his left leg consequent on a stumble in walking downstairs in December, 1883. This was attended with much effusion of blood, and subsequently there was evidence of thrombosis in some of the large veins of the same leg, with constitutional disturbance and loss of strength

which confined him to his room. After a temporary improvement there was a relapse with cough and loss of appetite and at last rather sudden death from syncope.

One of the most distinguished members of the profession, writing to a friend on hearing of the death of Mr. Cæsar Hawkins, said of him, "We have lost, I think, the clearest mind in our profession, in which accuracy was least swayed by imagination or temper or desire for renown. I have never known one more discreet or honest in council or less influenced by self-interest."[1]

*William James Erasmus Wilson*,[2] who was proud of his Scottish descent, was born in 1809, his father being then a medical officer in the navy. He received his earlier education at Dartford and Swanscombe, in Kent, and in 1825 he commenced his medical studies at St. Bartholomew's, where he is said to have been a favourite pupil of Abernethy. In 1831 he became a Member of the Royal College of Surgeons. Soon afterwards Dr. Jones Quain made him his assistant at University College, and subsequently he was appointed Demonstrator of Anatomy under Mr. Richard Quain. While holding this office he acquired repute as a skilful dissector and a successful teacher. In conjunction with Dr. Jones Quain he published a series of anatomical plates of the human body. In 1833 he published the 'Dissector's Manual,' a second edition of which appeared in 1853. In 1840 he was appointed Lecturer on Anatomy and Physiology at the Middlesex Hospital, and in the same year he published the 'Anatomist's Vade-Mecum,' illustrated by woodcuts. This was long a very popular manual and went through six editions, the last appearing in 1854. He was for some years a Consulting Surgeon to the Marylebone Infirmary, where he obtained much experience of general surgical practice, but ultimately he was led to adopt diseases of the skin as a special subject of study and practice. In the department of dermatology he worked with his cha-

[1] Obituary in the 'Lancet,' July 26th, 1884.

[2] 'Lancet,' and 'Brit. Med. Journal,' August 16th, 1884.

racteristic zeal and soon acquired a reputation which by degrees brought with it a large and very lucrative practice. In 1842 he published his 'Treatise on Diseases of the Skin,' a sixth edition of which appeared in 1867. In addition to this he published numerous other works, lectures, and papers on the skin and its diseases, the titles of which alone occupy a page and a half of our printed library catalogue. In 1843 he was elected a Fellow of the College of Surgeons, in 1870 a Member of the Council, and President of the Collège in 1881. In the same year he presided over the Dermatological Section of the International Medical Congress. In 1844 he was elected a Fellow of the Royal Society. He was elected a Fellow of this Society in 1839, in 1845 he served on the Library Committee, and two of his papers are published in our 'Transactions,' one "An Account of a Horn Developed from the Human Skin," vol. xxvii, the other "On the Echinococcus Hominis," vol. xxviii. Erasmus Wilson by his large professional income, but chiefly perhaps by his judicious investments, became possessed of great wealth, much of which he distributed during his lifetime with great but wisely discriminating liberality. The amount of his private beneficence, though known to be very large, cannot be accurately estimated, but he is well known to have been a most munificent public benefactor. Amongst other acts of munificence during his lifetime he expended £5000 in the endowment of a Chair of Dermatology in the College of Surgeons, and presented to the museum an extensive collection of models and drawings illustrative of diseases of the skin. In the University of Aberdeen, which had conferred on him the degree of LL.D., he founded, in memory of his father, a Chair of Pathology at a cost of £10,000. He contributed £10,000 towards the expense of bringing home the Egyptian obelisk which now adorns the Thames Embankment. In 1873 he restored the church of Swanscombe, in Kent. Besides contributing liberally towards the foundation of the Royal College of Music he endowed a Wilson scholarship at a

cost of £2500. In addition to large subscriptions to the Royal Medical Benevolent College he erected at his own sole expense a house for the head-master. He built a new wing and a chapel to the Sea-Bathing Infirmary at Margate, at a cost of more than £30,000; and as an eminent freemason he was a most liberal contributor to the various charitable institutions connected with the craft. In recognition of his professional eminence and his munificent public benefactions Her Majesty the Queen conferred on him the honour of knighthood in 1881.

Amongst Sir Erasmus Wilson's contributions to general literature may be mentioned, 'A Three Weeks' Scramble Through the Spas of Germany and Belgium,' published in 1858, and 'Cleopatra's Needle; with Brief Notes on Egypt and Egyptian Obelisks,' 1877.

About two years ago he was prostrated by a very serious illness which left him in delicate health and for more than a year before his death he had been totally blind. On July 23rd he was present at the consecration of St. Saviour's Church at Westgate-on-Sea, of which he laid the foundation-stone a year previously. A few days afterwards he was seized with inflammation of the bowels, which terminated fatally on August 7th. Sir Erasmus had no family but he leaves a widow and numerous deeply attached friends. Considering the benefits which by his great skill he conferred upon the numerous applicants for his professional aid and his munificent public and private benefactions he might with truth have appropriated the words of the patriarch, "The blessing of him that was ready to perish came upon me, and I caused the widow's heart to sing for joy."

*Mr. James Stock Daniel,* who died last August at the age of eighty, had been a Fellow of our Society since 1836. The son of a solicitor at Ramsgate he was educated at the Rochester Grammar School. He commenced his medical studies in Edinburgh, where he made the acquaintance of Thomas Wormald and Richard Owen, an acquaintance which was afterwards renewed at St. Bartholomew's.

There he acted as dresser to Wm. Lawrence, who so highly appreciated the brilliant social qualities of his pupil that he often asked him to take the bottom of the table at his dinner-parties. Amongst other distinguished men at St. Bartholomew's with whom Mr. Daniel formed a friendship which was continued through life were Sir George Burrows, the late Sir Thomas Watson, and Professor Richard Partridge. After leaving the hospital Mr. Daniel settled at Ramsgate, where for a number of years he had a large and lucrative practice. Amongst his intimate friends and patients there were Sir Moses Montefiore and the late Augustus Welby Pugin.

Mr. Daniel accompanied Sir Moses on two of his later journeys, viz. to Wallachia in 1866, and to St. Petersburg in 1872. The latter journey, on account of Sir Moses Montefiore's habit of rapid and continuous travelling, Mr. Daniel, although twenty years younger than his companion, found very fatiguing. He consequently had to stop on the way home and he never quite regained his former strength. He retired from practice as long ago as 1867. His friends report him to have been a most pleasant and popular man, well read and a good classic, with an extraordinary memory and brilliant powers of conversation. He was also a beautiful reader, rendering such pieces as "Twelfth Night," and the farce "A Fish out of Water," with wonderful effect.

*Dr. David Alexander King* having received his earlier education at the City of London School, was a highly distinguished student at St. Bartholomew's. In 1882 he graduated M.B. at the University of London, when he obtained the Scholarship and Gold Medal for Medicine, and was second with a Gold Medal in Obstetric Medicine. He served with distinction in various important offices at St. Bartholomew's, Casualty Physician, Assistant Demonstrator of Anatomy, House Physician and Ophthalmic House Surgeon, and he was appointed Assistant Physician to the Brompton Hospital for Consumption. He published in the 17th vol. of St. Bartholomew's Hospital

'Reports,' a paper "On Membranous Pharyngitis from Scarlatinal Infection" and in the same volume another paper on "Cases of Intestinal Obstruction." The 18th vol. contains an elaborate paper in which the results of seventy cases of typhoid fever are analysed, one result of the analysis being apparently to shew that although for various reasons the early administration of solid food during the convalescence from typhoid is injurious, yet that it is not the cause of the relapse which so often occurs.

In another paper published in 'Brain' (vol. v, p. 412) Dr. King has recorded a remarkable case of multiple cerebral tumours of unusual histological characters. All the papers afford evidence of accurate and industrious observation and close reasoning. His brilliant and highly promising career was cut short by consumption before he had completed his twenty-eighth year. He died at Torquay on September 4th, 1884.

*Dr. William Gillett Cory* was elected a Fellow of this Society in 1853. He obtained the license of the Society of Apothecaries in 1848, and graduated M.D. Paris in 1865. He commenced practice at Banstead, in Surrey. He afterwards practised at Brighton, then at Boulogne, and finally settled at Clifton, where, on the 25th September, 1884, he died in about two hours after an apoplectic seizure at the age of fifty-eight. In the 'Lancet,' 1848, he published a "Case of Placenta Previa successfully Treated by Removing the Placenta before the Child."

In the premature death of *Dr. Evan Buchanan Baxter*,[1] we have to mourn the loss of one who by his great ability, his varied learning, and yet more by his admirable character was known by his many friends, and in the course of time would have been recognised by the entire profession and by the public as presenting the very highest type of an able and accomplished physician.

His father, James Baxter, a personal friend of Chris-

[1] See an interesting memoir of Dr. Baxter by one of his colleagues, 'Lancet,' January 24th, 1885.

topher North, was descended from an old Scotch Presbyterian family. In early manhood he went to St. Petersburg, where he was Director of the English School in that city, and there he married Miss Ross, the daughter of a Scotch merchant who had settled in St. Petersburg. Of this marriage a daughter who died in infancy and the subject of our notice, who was born in 1844, were the only children. The mother died of consumption while her son was still very young. Soon after the death of his wife Mr. James Baxter was appointed Government Inspector of Schools in Russian Poland and went to reside at Kaminetz in the province of Podolsk.

There until the age of sixteen young Baxter was brought up and educated by his father and an old French tutor, and there, with a great natural aptitude for linguistic acquirements, he was placed in favorable circumstances for obtaining a practical knowledge of the chief European languages. Russian and German were the languages of the people amongst whom he was brought up, English was the language of his home, French he learnt from his tutor, and Latin from his father. In October, 1861, he came to England and entered the General Literature and Science Department of King's College. At that time he knew nothing of Greek, but in the course of the next year he had acquired such a knowledge of Greek literature and history as enabled him to obtain an open scholarship in classics at Lincoln College, Oxford, where he remained for three terms. His intention then was to become a classical tutor and Fellow and without doubt he would have obtained the object of his ambition; but "there's a divinity that shapes our ends, rough-hew them how we will." Baxter's university career was cut short by a summons to attend his father, who was dangerously ill in Russia and with whom he remained until his death, which occurred at the end of a year. On returning to England he found that he could not resume his place at Oxford, for his scholarship had lapsed, and for other reasons he decided to enter the profession of medicine, which held

out, as he said, "an opportunity for the study of physical science and a hope of comparative intellectual freedom." In 1864 he entered the medical department of King's College, where he obtained the first Warneford entrance scholarship and in the following spring he was elected a junior medical scholar in conjunction with the late Prof. A. H. Garrod and Prof. Curnow. In 1865 he obtained the Dasent prize open to the whole College and usually won by students in general literature, the subject for the essay being "The Minor Poems of Milton."

In 1865 he matriculated in the Honours division of the University of London, and in 1869, when he graduated M.B., he obtained the scholarship in medicine and was second with a Gold Medal in Obstetric Medicine. In 1870 he obtained the degree of M.D. and was marked with a star as worthy of the Gold Medal.

He held various appointments at King's College. In 1868-9 he was House Physician. For two years, in 1870-71, he was Sambrooke Medical Registrar, and he discharged the duties of this office in so exemplary a manner as to earn the gratitude alike of students and physicians, of the former especially, by his admirable bedside teaching. In 1871 he was appointed Medical Tutor and after conscientiously discharging the duties of this laborious office for three years, he was chosen to succeed Dr. Garrod as Professor of Materia Medica and Therapeutics and at the same time he became Assistant Physician to the hospital. On resigning the office of Medical Tutor he was elected an Honorary Fellow of King's College. In 1872 he became a Member of the Royal College of Physicians and in 1877 he was elected a Fellow. He was subsequently appointed an examiner in Materia Medica and Therapeutics at the College, and he held for five years the same office in the University of London. Whilst still Medical Tutor he was elected on the medical staff of the recently founded Evelina Hospital for Sick Children, and he worked diligently at the diseases of children, first in the out-patient rooms and then in the wards, for the next nine or ten

years. During the same period he made his well-known admirable translation of Rindfleisch's 'Pathological Histology' for the New Sydenham Society, and he revised the fourth edition of Garrod's 'Essentials of Materia Medica.'

In the 'Practitioner' of 1873 he published a valuable paper entitled "The action of Cinchona Alkaloids and their Congeners on Bacteria and Colourless Blood-corpuscles." He was subsequently asked by the medical officer of the Local Government Board to investigate the value of disinfectants, the result of which inquiry was an admirable essay, published in a Blue-book in 1875, under the title "Reports on Experimental Study of Certain Disinfectants."

In 1876 he published in the 'British and Foreign Medico-Chirurgical Review' an able summary of the physiology of the vaso-motor nervous system, in which he showed a thorough knowledge of the history and position of that important subject.

His accurate and extensive knowledge of skin diseases, derived from many years' work at the Blackfriars Hospital, is displayed in a paper on "General Exfoliative Dermatitis," published in the 'British Medical Journal,' vol. ii, 1879.

He had for several years contributed to the 'Academy' a series of "Physiological Notes." In March, 1880, he published, in conjunction with Dr. Willcocks, a paper on "Clinical Hæmometry," and his latest literary work was a brief note in 'Brain' (January, 1884) welcoming the publication of a Russian review devoted entirely to neurology. In 1881 Dr. Baxter was appointed Physician to the Royal Free Hospital, with the charge of instructing the lady pupils in clinical medicine, a work which he performed with characteristic zeal and efficacy.

Dr. Baxter had always been delicate, and during the last two years of his life he had a succession of serious illnesses, which were borne with heroic fortitude and even cheerfulness. First he had an attack of pleurisy in the right side, then a similar attack of the left. With

this there was some obscure intestinal trouble. Subsequently disease, probably tuberculous, attacked the apices of both lungs, and lastly came albuminuria and dropsy with exhausting diarrhœa, ending fatally on the 14th of January. He leaves a widow and a host of devoted friends to mourn their irreparable loss.

Those qualities of Dr. Baxter's mind and character which, in addition to his powerful and highly cultured intellect, excited the love and admiration of his friends, may briefly be said to have been his conscientious devotion to every work and duty which he undertook, his scrupulous accuracy of statement, his remarkable power of clothing his thoughts in clear, vigorous, and appropriate language, his fine sense of humour, and with all this his charming modesty of demeanour and his detestation of unseemly self-assertion and display. He had been a Fellow of this Society since 1874 and a Referee since 1881.

*Mr. William Collins Worthington* was born at the commencement of the century (February 26th, 1800) and at an early age began the study of medicine and surgery as a resident pupil at the Norwich County Hospital. There he had for his teachers the late Drs. Rigby and Philip Martineau, from whom he imbibed a love of his profession and a special taste for surgery, together with such instruction and experience as made him in after-life a successful lithotomist. On leaving the Norwich Hospital he entered at the Middlesex Hospital, where he worked under Sir Charles Bell, and studied anatomy at the school of Mr. Joshua Brook. He became a Member of the Royal College of Surgeons in 1819, and an Honorary Fellow in 1844. In 1822 he commenced practice at Lowestoft, and he soon established a cottage hospital, which under his auspices has grown to its present dimensions, with accommodation for thirty beds.

He was actively and extensively engaged in practice for fifty years, during which time he was a frequent contributor to medical literature. He was a diligent student of

pathology and never lost an opportunity of investigating disease by post-mortem examinations. He thus collected a considerable number of interesting pathological specimens. Mr. Worthington had been a non-resident Fellow of the Society since 1842, and he contributed three papers to the 'Transactions,' one on "Stricture of the Trachea" (vol. xxv); one on "Fistulous Communication between the Bladder and Ileum simulating Stone" (vol. xxvii), and a third on "A Case of Sacculated Œsophagus" (vol. xxx). He also published papers in the 'Lancet' on "Aneurism," "Paracentesis," and other subjects. Although not physically a strong man he had nearly completed his eighty-fifth year when he died on the 31st January last. This prolongation of his life with his mental faculties unimpaired he attributed to his strictly abstemious habits. He was always opposed to the dietetic use of alcoholic stimulants, so that both by example and by precept he was an influential promulgator of temperance doctrines. Mr. Worthington was highly respected and esteemed by all classes of the community, amongst whom his long, useful, and most honorable life had been passed.

It will have been seen from the report of the Council that our merely financial losses by deaths, resignations, and non-payment of subscriptions, have been considerably more than counterbalanced by the unusual number of new Fellows elected, and the balance-sheet shows a satisfactory excess of income over our ordinary annual expenditure.

The Council confidently appeal to the Society for their cordial approval of the extraordinary expenditure which has been incurred for the removal of the very grave sanitary defects which were found to exist in the basement of our building, defects of so serious a character as not only to imperil the health of those who reside on the premises, but also, though in a less degree, that of the numerous members of our own and other Societies who meet within these walls, and who have a right to expect that all due care shall be taken to guard them against the dangers resulting from defective drainage.

But in order to render our premises entirely suitable for the important work of various kinds which is here carried on, something is yet required. The lighting and ventilation of this meeting-room are not satisfactory; it is very difficult to maintain a pleasant uniform temperature; and towards the termination of a full meeting the air becomes not only unpleasant but positively unwholesome. There is also abundant evidence that the products of gas combustion are destroying the bindings of our extensive and valuable library. The Council whom you elect to-day will of necessity have their attention directed to this very important question, and if their deliberations should result in making as great an improvement in the lighting and ventilation as has lately been effected in the drainage, they will deserve and will doubtless receive the thanks of the Society.

In conclusion, I think it will be admitted by all who have attended to the work of the Society, that the papers and discussions during the past year have been at least equal to the average of former years in interest and importance, and an inspection of the list of papers to be read will show that we have an abundance of good material to occupy us during the remainder of the session.

The subject of cholera has for some time past excited much interest, and this interest increases as the time approaches when a reappearance of the disease in Europe is not improbable. It has therefore been thought desirable that the etiology, pathology, and treatment of cholera should be discussed at an early meeting of the Society, and I have undertaken to initiate such a discussion on the 24th inst., when it is hoped that we may have a large attendance and an instructive debate.

# CASE

OF

# CIRSOID ANEURISM ON THE DORSUM OF THE FOOT,

## WITH REMARKS ON THE DISEASE.

BY

WALTER EDMUNDS, M.C.

Received April 7th—Read October 28th, 1884.

JOHN B—, æt. 29, was admitted into St. Thomas' Home for a swelling on the dorsum of the left foot. The tumour was round, about one and a half inches in diameter, pulsated with the pulse, and expanded in all directions; there was a well-marked thrill with the pulsation. Pressure on the anterior tibial artery only diminished the pulsation, but compression of both the anterior and posterior tibials completely arrested it. This is not a matter of course for there might have been a communication with the anterior peroneal artery.

The swelling had been noticed six weeks. There was no history of injury, or of syphilis. After a few days' rest in bed an Esmarch's bandage was applied firmly above and below, but lightly over, the aneurism. This was kept on for an hour and fifty minutes; on removing the bandage the pulsation returned and no improvement had been produced. It was then decided to excise the aneurism. Accordingly a

week later, the patient being anæsthetised, a longitudinal incision was made over the tumour. The sac was exposed, and on its lower side two arteries proceeding from it were found: they were each tied by two ligatures and the vessels divided between them. The artery which supplied the tumour from above was probably the dorsalis pedis. To have exposed it for ligaturing at a safe distance above the aneurism would have necessitated dividing the annular ligament; the anterior tibial was therefore exposed above the ankle, tied in two places and divided between the ligatures. This vessel was enlarged. The aneurism still continued to pulsate, the dorsalis pedis was then tied immediately above the sac and divided, and the sac dissected out, the vessels being tied as they were exposed: altogether seven fair-sized arteries were found communicating with the aneurism. All vessels were tied with catgut. There was some absorption of the tarsal bones lying immediately under the aneurism, but fortunately no joint had been opened.

The wound healed slowly but completely, and the patient went out well.

On examining the tumour it was seen that except for one bend on its position surface the aneurism was a simple sac with numerous communicating vessels. The specimen is in the museum of St. Thomas's Hospital.

*Remarks.*—The dorsalis pedis artery is peculiarly exposed to injury both directly from blows, and indirectly from sprains of the foot, traumatic aneurisms of this artery are consequently not uncommon; but spontaneous aneurism is rare, and I have only succeeded in finding records of six cases in which no cause could be assigned. An abstract of these cases is added.

With respect to the anastomotic nature of the aneurism it would seem that Professor Gross's case (*vide infra*) was also of this kind, for he remarks that "from the great number of ligatures required to arrest the bleeding it would appear that the tumour was somewhat of the character of an aneurism by anastomosis." A case is recorded too by Fleury, in which a punctured wound of one of the smaller arteries

of the dorsum of the foot was followed by a tumour which appeared to consist "of little vessels anastomosing among themselves to infinity."[1]

Mr. Poland had a case under his care in which there was a cirsoid aneurism on both the plantar and dorsal aspect of the foot,[2] and a somewhat similar case is recorded by Nicoladoni.[3]

The treatment of aneurism of the dorsalis pedis or its branches must depend on the exact nature of the case.

The patient in the Navy (No. 1 in appendix) was cured by pressure, and if the aneurism can be completely controlled without any great amount of pressure, and if it be not freely expansile in all directions a trial may be made of digital compression or, if this cannot be arranged, of instrumental compression or of Esmarch's bandage. These methods having failed or being thought inapplicable we have to choose between the various operative procedures.

One of M. Panas's cases (No. 6) was cured by the injection of a solution of perchloride of iron, and this is the treatment recommended by M. Henri Toussaint in his able thesis on this disease.[4] There is, however, danger of inflammation of the sac in this method, and the proximity of the tarsal joints is a special reason for avoiding this complication. Simple ligature of the dorsalis pedis may fail, though it succeeded in Mr. Savory's case (No. 5), and the surest treatment would seem to be excision of the aneurism. Even a case like Mr. Adams's, in which the aneurism had in all probability opened into a tarsal joint before the patient came under observation, would not be an objection to that treatment, for the opening into the joint would be exposed in the operation, and the foot could be, if it were thought necessary, amputated, and this, indeed, is the treatment which eventually had to be adopted in that

[1] 'Archives générales de Méd.,' 3me série, tome v, 1839.

[2] 'Lancet,' vol. i, 1866, p. 536.

[3] 'Archiv für klin. Chir.,' Band xviii, 1875.

[4] 'Considérations sur l'anatomie de l'artère pédieuse et sur ses anévrysmes, par Henri Toussaint. Paris, 1879.

case. In fact, the treatment recommended by Sir Astley Cooper in his lectures on Surgery is still the best. Speaking of aneurism of the anterior tibial artery, he says: "Mr. Henry Cline had a case of this disease upon the upper part of the foot, and he tied the anterior tibial at the lower part of the leg, but the pulsation in the aneurism continued when the boy quitted the hospital. It will be, therefore, right to tie the artery by opening the sac, so as to secure it above and below the aperture, if the aneurism be seated low down in the limb, as the anastomosis with the plantar arteries is exceedingly free." [1]

It would be well, however, to bear in mind the possibility of finding implication of the tarsal joints, and to be provided in doubtful cases before commencing the operation with permission to amputate should that be thought advisable.

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## Abstract of Six previously recorded Cases of Spontaneous Aneurism of Dorsalis Pedis Artery.

1. *The case in the Navy* ('Report on Health of Navy,' 1872).

Patient was a stoker R.N. He had an aneurism on the dorsalis pedis artery, which was cured by compression at Haslar Hospital.

2. *M. Panas's first case* ('Bulletin Soc. de Chirurgie,' 3me série, tome ii, 1873).

Labourer, æt. 58, spontaneous aneurism of right dorsalis pedis. The tumour was partly beneath and partly below the annular ligament; the skin was inflamed. Before admission the aneurism was mistaken for an abscess, but on commencing to incise it the pulsations were noticed and the incision was not continued. Three days later slight oozing, and in three more sharp hæmorrhage occurred from the wound. To arrest this M. Panas tied the anterior tibial, and this being insufficient also the dorsalis pedis below the aneurism. This stopped the hæmorrhage and for a time the pulsation, though it subsequently recurred. The surface of the tumour sloughed,

[1] 'Lectures on the Principles and Practice of Surgery,' vol. ii, p. 63.

rigors came on, and the patient died of pyæmia with abscesses in the lungs.

The treatment M. Panas would have adopted in this case had not the hæmorrhage rendered ligature necessary was the injection of perchloride of iron with temporary pressure round the aneurism.

3. *Professor Gross's case* ('Philadelphia Medical Times,' 1874).

Coloured man, æt. 59. There was an aneurism the size of a small bird's egg on the external aspect of the dorsum of the left foot, first noticed four months before patient was seen; no history of injury. As the aneurism was external to the position of the dorsalis pedis artery it was thought to be on one of its branches.

*Treatment.*—Under ether the femoral was controlled and the aneurismal sac laid open. "It seems impossible to separate the sac and trace out the artery by which it is supplied, and hence it becomes necessary to ligate the anterior tibial which is accomplished by extending the incision upward and passing a ligature round the vessel just above the ankle, where it lies between the tendon of the extensor longus digitorum and the extensor proprius pollicis muscles. There is still copious hæmorrhage proceeding from the recurrent circulation, which is dependent on the perforating branches of the plantar arteries, and from the great number of ligatures required to arrest the bleeding it would appear that the tumour has somewhat of the nature of an aneurism by anastomosis."

Secondary hæmorrhage occurred a week later and was stopped by acupressure and the ligature of the anterior tibial a second time (higher up). Hæmorrhage did not recur, but the patient died a fortnight after from pyæmia with suppuration in the ankle-joint. The anterior tibial artery was found at the post-mortem to be ossified.

4. *Mr. Adams's case* ('British Medical Journal,' vol. ii, 1877, p. 804).

A labourer, æt. 29, no known cause, never had syphilis. On dorsum of foot over outer side of astragalo-scaphoid articulation a tense, shining pulsating swelling the size of a walnut, diagnosed to be an aneurism. It was thought better to tie the anterior tibial high up in the leg rather than near the aneurism for fear of finding the vessel diseased and creating suppuration among the tendons. Pulsation ceased. A fortnight later the sac had not consolidated and was opened. A week later suppuration was found to exist in astragalo-scaphoid joint and subsequently spread to other joints necessitating amputation of foot, after which patient recovered.

5. *Mr. Savory's case* ('British Medical Journal,' vol. i, 1878, p. 75).

A man, æt. 49. Aneurism on the dorsalis pedis behind the angle formed by the first and second metatarsal bones. All pulsation arrested by compression of the artery above the tumour. Compression being unsuccessful the dorsalis pedis was ligatured an inch and a half above the tumour, all pulsation ceased, but in three minutes slight pulsation reappeared, and some light pressure was made on the sac. The aneurism was cured.

6. *M. Panas's second case* (Thesis by Henri Toussaint, 1879).

Male, æt. 38, no history of injury, doubtful history of syphilis. Pulsating tumour the size of a large nut on the dorsum of left foot.

Treated, firstly, by application of Esmarch's bandage but without result.

Secondly, by electro-puncture but no clot formed and tumour continued to pulsate.

Thirdly, by mechanical compression; no benefit.

Fourthly, by injection of perchloride of iron into sac, the femoral and the dorsalis pedis (below aneurism) being compressed and an Esmarch's tube tightly applied round the lower part of the thigh. This completely cured the aneurism.

(For discussion on this paper see 'Proceedings of Royal Medical and Chirurgical Society,' New Series, vol. i, p. 283.)

ON

# EXPIRATORY CERVICAL EMPHYSEMA,

THAT IS,

## EMPHYSEMA OF THE NECK OCCURRING DURING LABOUR AND DURING VIOLENT EXPIRATORY EFFORTS.

### AN EXPERIMENTAL INQUIRY.

BY

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(Received April 16th—Read November 11th, 1884.)

In spite of the fact that emphysema of the neck, face, and other adjacent parts is an accident of labour which is not extremely rare, its pathology has hitherto been the subject of various hypotheses, amounting to little more than guesses, and even at the present time has never, hitherto, been the subject of accurate investigation.

The present paper is an effort to place the pathology of this affection on a firmer basis, and the investigations here detailed suggested themselves by way of corollary to experiments concerning the subject of artificial respiration in newborn children and kindred subjects related in the 'Med.-Chir. Transactions,' vol. lxiv, 1881, pp. 41—101, and vol. lxv, 1882, pp. 75—86. But the latter

investigation, concerning mediastinal emphysema and pneumothorax after tracheotomy (in which the route followed by the air was proved to be from the tracheotomy wound, beneath the deep cervical fascia and so into the tissue of the anterior mediastinum beneath the pleura), suggested that emphysema of the neck during labour might follow the same route in an opposite direction. This it was determined to test by experiment on the fœtus, for the reasons that although it is true that the affection does not concern fœtuses but adult women, yet (1) fresh fœtuses have usually fairly healthy lungs; (2) their nearly identical age makes them a nearly uniform material; (3) the experiments could in any case only be illustrative, and therefore could only possess value in proportion to the amount of correspondence which they showed to the known phenomena. In this way adult subjects could only serve in the same manner as fœtuses. With these considerations it was determined to choose fœtuses as the subjects of experiment. Two children only who had breathed for a very short time (Experiments 9 and 10) were also used.

*Frequency of occurrence of emphysema.—Johnston and Sinclair*[1] (p. 517) had seven cases in the course of seven years (13,748 labours) in the Dublin Hospital, or less than 1 in 2000 cases.

*Ætiology.*—It is generally agreed that emphysema of the neck occurring during labour is due to bearing down. This will be seen on reference to almost any case on record (see table of references). Thus in the Dublin Hospital all the patients affected were primiparæ, and in all the cases severe bearing-down efforts are noted; it is obvious that any cause of obstruction to labour must so far favour it.

The time of its occurrence tells the same story. It never occurs before the second stage of labour, though it may not show itself till the third stage or altogether after labour (see *Dunn's* first case) In any case, how-

[1] For the list of works quoted in this paper see p. 67.

ever, it is no doubt produced during the second stage, even if it does not appear till later.

It need not occur, however, in connection with parturition at all, but may follow violent coughing (as in *Roché's* case, in which there was a foreign body in the trachea), or perhaps straining at stool (see remarks by *Dr. Otis* on *McLane's* case). Indeed the bearing down in labour has nothing special about it except that straining is maintained for a longer time than under other conditions.

*Clinical course.*—It will perhaps be well to quote one or two recent cases. Those of *Dunn* and *McLane* are selected as amongst the latest recorded.

*Dunn* records four cases of emphysema during labour; with two of which no details are, however, given. These are obviously cases of emphysema beginning below, and probably in connection with rupture of the uterus. Both ended fatally. This subject is alien to the present inquiry. The other two cases are more to our purpose.

(1) The first of these occurred in a primipara, who had an "ordinary labour" (whatever that may mean). During a violent effort to expel the placenta the right side of the face became swollen and crepitant. (N.B.—It came out in the discussion that the swelling was first noticed by the patient, and not till half an hour after the expulsion of the placenta, which throws considerable doubt on the time of the occurrence.) The swelling seemed to begin on the lower part of the right submaxillary region, and extended upwards on the nose and cheek as far as the zygoma; it gradually extended to the neck; no other symptoms were noticed. After some hours, the swelling, which was puffy and moveable, extended to the upper side of the chest and right arm. There was no cough, pain, or dyspnœa, and no change in the respiration. In the next two days the emphysema extended lower on the chest. The air remained four days and then gradually disappeared.

(2) The emphysema in the second case began (?) on the upper part of the right side of the chest under the

clavicle, and extended upwards as far as the zygoma and down to the middle third of the chest; also to the shoulder and upper part of the arm.

The details are so meagre that the order of the appearance of the emphysema is open to considerable doubt, but the localities affected may be accepted as correct.

*McLane's* case was that of a primipara, æt. 21. The first stage was very painful, the pains being long and very severe, lasting two hours, and necessitating the use of chloroform.

During the second stage there was violent straining; during one of these efforts the face became congested and purple, a swelling appeared on the right side near the trachea, and became much larger during the next three or four pains, which followed each other rapidly. The swelling extended to the right cheek. As soon as the patient recovered from the chloroform she complained of a constriction in the throat, and had some difficulty in swallowing. "Her neck, previously slender, was now thick and œdematous; her face so puffed that on the right side the eyelids were closed, as if by dropsical effusion, and the features effaced." The swelling crackled on touch. There was no emphysema below the clavicle. The whole swelling had disappeared in a week.

We learn from this and other cases that the swelling appears during the second stage of labour, generally during some violent straining effort, that it first appears in or about the suprasternal notch, extends rapidly upwards along the neck, and may reach the face; it may also extend down over the chest and down the arms. Its course reminds us of that of extravasation of urine affecting the opposite sex in a different part of the body, except that in the latter case the urine is unable to travel down the limbs. To trace the possible course of the air or the urine would be waste of time, since air easily travels all over the surface of the body in the subcutaneous cellular tissue—a fact practically useful in the skinning of animals—and urine for its part may do the same. All varieties

of its extension may be found in the appended references. The other points of interest are that the air is all absorbed in a week or so, without any ill-consequences, and never ends fatally (a fact partly explaining the doubt as to its pathology).

The last, but perhaps most important, fact is that there is no disorder of respiration, and *never pneumothorax.*

*Pathology.*—We now come to the chief subject of our investigation.

The theories to account for the phenomena are various.

*Cloquet* (p. 33) gives a good account of a case which of course recovered. He says, "It would be impossible for me to define here precisely in what spot the bronchi or their branches were ruptured."

*Ménière* quotes Cloquet.

*Blundell* (p. 473) attributes it to "*rupture of the trachea and bronchi.*" He saw it twice in the same patient, a vociferous Hibernian, and in no other case.

*Depaul* (p. 689) gives two post-mortem accounts in which there was interlobular emphysema of the lung, but not of the neck.

*Watson* (vol. ii, p. 176) says, "Air passes into the mediastinum, and so into the neck."

*Johnston and Sinclair* do not explain it.

*Roché* (p. 252) in a case in which a foreign body had got into the trachea, found air in the mediastinum, and double interlobular emphysema, especially on the left side.

*Soyre* refers to Cloquet's case, and says, "The *rupture of the trachea* was situated a little above the bifurcation of the bronchi."

*Oppolzer* (p. 582) says that the commonest site of interlobular emphysema is the anterior edges of the upper lobes. "Serious consequences may ensue, if the air advances towards the hilum of the lung and the mediastinum, and from thence ascends into the subcutaneous cellular tissue of the neck and face. This is, however, on the whole a rare event."

*Traube* (p. 89) quotes Roché.

*Mackenzie* (p. 205) quotes Watson.

*Whitney* (p. 350) relates a case, but does not speculate on the pathology.

*Haultcœur* (p. 420) refers to Depaul.

*Schroeder* (p. 455) simply says it is due to "rupture of the air-vesicles."

*Prince* relates a case only.

*Worthington* does the same.

*Atthill* refers to Depaul.

*Alexeef* refers to Depaul and Haultcœur.

*Nelson* relates a case.

*Spiegelberg* (p. 419) mentions it only as an evidence of the force of bearing down.

*Dunn* (p. 397) remarks that in his second case interlobular emphysema due to laceration of the air-vessels or of the bronchial tissue was possible. This rupture generally causes emphysema of the pleura, which may extend to the subcutaneous tissue of the thorax and body generally. He remarks that Jones and Sieveking say that the same condition may arise from laceration of the trachea. I have repeatedly searched for this reference but failed to find it.

*McLane* (p. 582) believed the lesion in his case to have been rupture of the trachea. He had seen but one other case, and in that there was rupture of the pulmonary vesicles.

We have thus a variety of theories:

(1) Rupture of the bronchi.

(2) Rupture of the trachea.

(3) Rupture of the lung.

The *post-mortem records* bearing at all on the subject are meagre in the extreme; they comprise two autopsies, showing interlobular emphysema of the lung, but not of the neck (Depaul), one case of interlobular emphysema, and air in the mediastinum (Roché); the other theories are not supported by any actual observations that I have been able to find.

It became necessary then to put these theories to the test.

*Mode of experiment.*—It was obviously impossible to exactly imitate bearing down, but on considering the conditions it appeared that they would be best imitated by putting pressure on the lungs filled with air and within the thorax. If any encouraging results followed it would then be advisable to study the behaviour of the lungs removed from the chest, so as to estimate the influence of the thoracic box.

The desideratum was *to produce emphysema of the neck without pneumothorax.* If pneumothorax should occur in some cases and not in others it would remain to eliminate its influence by a consideration of the cases.

*Apparatus.*—The apparatus consisted of a simple mercury manometer marked in millimetres, attached to a tube ending in a tracheal cannula. A T-piece answered the purpose of a mouth-piece for the operator, connected on one side with the manometer, and on the other with the trachea of the fœtus through the tracheal cannula.

Some experiments were performed with the lungs *in situ*, others with the thorax open, others with the lungs removed from the chest. In one experiment an attempt was made to trace the course of the air in former experiments by means of a coloured gelatine mass.

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Experiment 1.—March 24th, 1882. Male child, born March 23rd.

Experiment March 24th, 3 p.m. (about thirty-six hours). Tracheotomy was performed, a cannula tied into the trachea and connected with a V tube filled with mercury, to the india-rubber tube of which was connected a T-piece, through which inflation was practised.

On blowing through the T-piece the chest expanded, then two small projections appeared below the third rib on each side in the nipple line; shortly afterwards the skin along the right sterno-mastoid became swollen; the

swelling was tympanitic; on pressure air escaped from the tracheotomy wound.

The extreme height of the manometer = 50 mm., *i. e.* height of mercurial column = 100 mm.

*Autopsy.*—Thorax opened under water. Double pneumothorax. Air bubbles beneath pulmonary pleura at the reflection of the pleura from the root of the right lung over the anterior mediastinum. Some diffuse subpleural emphysema.

On inflating the lungs again after opening the thorax, air distends the reflection of the pleura over the root of the right lung still more, and on the left side in the same manner, raising the pleura from the pericardium, and following the whole course of both phrenic nerves, dissecting up the pleura.

On plunging the fœtus again under water and blowing, air escapes from two small holes in the anterior inferior part of each lung, but in larger bubbles from the reflection of the pleura over the root of the right lung (? were the small holes in the lungs punctures or ruptures).

The region of the cervical emphysema was next dissected; the air was found along the course of the right internal jugular vein:

(*a*) A bubble could be pressed into this collection from that in the anterior mediastinum.

(*b*) A bubble could be pressed the reverse way.

The air seemed to have passed from the anterior surface of the right lung—near and in front of its root—into the anterior mediastinum, behind the vena cava superior, and so along the right internal jugular vein.

*Remarks.*—The object of the experiment was to see in what direction air would escape from the lungs if overblown. This succeeded, the air passing from the lung substance near the root, behind the pleura, and along the great vessels of the neck.

Pneumothorax was, however, produced on both sides.

EXPERIMENT 2.—March 31st, 1882. Fœtus born March 29th, after placenta prævia (? full time).

Experiment March 31st, 3 p.m. Tracheotomy was performed and a cannula tied into the trachea as before.

Inflation was then practised intermittently, so as to imitate, as far as possible, bearing down during labour.

The maximum height of the mercury column = 60 mm., *i. e.* whole column = 120 mm.

*Autopsy.*—On opening the thorax, air escaped from both pleuræ. Universal subpleural emphysema. Air occupies the anterior mediastinum and spreads in front of the root of each lung into the anterior mediastinum. No emphysema in right side of neck. Air extends along the course of the left internal jugular vein and can be pressed into the collection in front of the root of the right lung, to and fro; it passes under the left innominate vein.

On reinflating the lungs, air is distinctly seen to pass from beneath the pleuræ near the root of the right lung, in front of the right lung into the anterior mediastinum. Similarly on the left side, where the air chose the same path upwards along the left phrenic nerve. Air escaped from several ruptures in the pulmonary pleuræ.

*Remarks.*—The object and results practically the same as in Exp. 1.

EXPERIMENT 3.—April 4th, 1882. Full-time male child, born after turning, April 2nd, 2 a.m.

Experiment April 4th, 3 p.m. An incision was made from the ensiform cartilage to the pubes. Part of the right fourth rib was excised, the pleura exposed, and a cannula tied into the pleural sac, and then connected with the manometer as before.

Inflation was then practised, and the mercurial column raised to 50 mm., *i. e.* the column = 100 mm. in height; at this height air escaped through a hole made in dissection at the junction of the diaphragm and right lower

ribs, wounding the diaphragm but not the pleura, but leaving the pleura unsupported and therefore weak at this point; from this spot the air travelled behind the peritoneum, stripping it up. The cannula was quite secure. On the left side the same condition was found.

*Remarks.*—The object was to compare the strength of the lung and pleura, or rather of the lung and pulmonary pleura, with that of the parietal pleura. The experiment failed, but it showed the extreme ease with which air travels beneath the pleura and peritoneum.

Experiment 4.—April 5th, 1882. Full-time male child, born April 2nd, 1882.

Experiment April 5th, at noon. The same apparatus was used as before, except that the india-rubber tube, instead of passing directly to the tracheal cannula, passed to a Wolff's bottle filled with a warm solution of coloured gelatine, which was therefore to be driven into the lungs.

On blowing at the T-piece the gelatine solution passed into the lungs.

The maximum height of the mercurial column = 30 mm., *i. e.* the height of the whole column = 60 mm.

*Autopsy.*—Coloured gelatine in both pleuræ. The pleuræ universally œdematous with the same fluid, but most has collected in front of the root of each lung, in the anterior mediastinum, but not along the great vessels of the neck. On blowing air through the tracheal tube, air escapes and forms emphysema on the front of the left lung in one place, and on the front of the root of the right lung, but in no other part. The most œdematous parts are perfectly air-tight.

*Remarks.*—The object was to produce, if possible, emphysema of the neck with some coagulable injection, to allow of a leisurely dissection. The gelatine, however, simply transuded almost as if through a filter. I had observed this in the case of water in an experiment not here recorded. The most remarkable fact is that the

lungs are simple filters to fluid when they are still absolutely air-tight. The bearing of this is wide, but need not here be enlarged upon.

EXPERIMENT 5.—April 5th, 1882. Female child, born April 13th, weight 4 lbs., length 17½ in.

N.B.—Manometer was broken at the beginning of the experiment and could not be used.

Tracheotomy, cannula tied into trachea. Lungs inflated intermittently to imitate intermittent bearing down during labour.

*Autopsy.*—Double pneumothorax.

General subpleural emphysema, *especially in front of both roots of the lungs;* no emphysema of the neck.

On inflating the lungs again, air slightly escaped from a few places.

*Remarks.*—In the absence of the manometer an attempt was made to repeat the production of emphysema of the neck, but without success. The fœtus was probably immature.

EXPERIMENT 6.—April 17th, 1882. Male child, born April 15th, 9 a.m. (hand presented), full time. Length 20½ inches, weight 7 lbs.

Experiment April 17th, 11 a.m., *i. e.* fifty hours after death, continued for an hour and a half. Tracheotomy, cannula tied into trachea, inflation intermittent.

An escape of air from the incision was noted, ? from trachea. Cannula was tied in again.

Escape of air continued. The skin incision was prolonged to the sternum, the trachea was opened as low as possible and the cannula tied in again.

Escape of air continued, apparently not from trachea but from right side of it, in the region of the large vessels.

N.B. The escape of air is only occasional, and does not prevent the mercurial column from rising to 40 mm. (*i.e.* column=80 mm.).

*Autopsy.*—Double pneumothorax.

Both lungs show subpleural emphysema, the right more than the left.

A large collection of air-bubbles existed on either side in front of the root of each lung, the air having got beneath the reflection of the visceral pleura over the mediastinum nearly everywhere on the right side. Another collection of bubbles on each side where the phrenic nerves reach the diaphragm. A large collection in front of the base of the right lung. The *right* half of the anterior mediastinum is one huge bubble; there are no bubbles on the left half.

The right side of the neck was very carefully dissected.

Air is present beneath right sterno-mastoid muscle, corresponding to the place from which it escaped during the experiment. A continuous chain of air-bubbles extends from the anterior mediastinum to the collection along the vessels of the neck on the right side, following the course of the phrenic nerve. Air can be pressed from collections on the upper part of the diaphragm, along the course of each phrenic nerve to the collection in front of the root of each lung.

On the left side a few small bubbles are found in the neighbourhood of the incision, and seem to have come from the incision.

On reinflating the lungs, air can be seen to pass behind the pleural reflection into the mediastinum as above, and to escape freely from the surface of both lungs.

*Remarks.*—The object was still the same, and the experiment succeeded in showing plainly the route taken by the air from the lung to the neck, but pneumothorax was also produced

EXPERIMENT 7.—June 26th, 1882. Small fœtus, obviously premature, born June 24th.

Experiment June 26th, 5 p.m. Weight of fœtus 2¾ lbs., length 15½ inches. Tracheotomy, cannula tied into trachea. Intermittent inflation. Maximum rise of mercurial column=40 mm. (*i. e.* height of whole column=80

mm.) Air escaped eventually from the right side of the incision.

*Autopsy.*—On opening the chest one or two doubtful bubbles escape from the right pleural sac, *none from the left.*

Anterior mediastinum airless in most parts. Along the great vessels on both sides (especially the right) there is a continuous chain of air-bubbles, passing behind the innominate vein towards a collection in front of the root of each lung.

Subpleural emphysema (slight) in one or two patches in front of each lung, but especially on their inner surface near the root.

*Remarks.*—The object was the same as that of the preceding experiments, and the result very instructive. On the right side the air had taken the usual route, but there was a little doubtful pneumothorax. On the left side, however, the air had passed into the neck without escaping into the pleural sac.

EXPERIMENT 8.—November 3, 1883. Male, full-time fœtus, quite fresh.

Experiment at 3 p.m. Thoracic viscera with the trachea were removed *en masse.* The lungs showed a few superficial lobules expanded, mostly at the apices.

The trachea was attached to the manometer, and a looking-glass arranged so as to show the side of the lungs away from the experimenter.

The pressure was begun at a rise of 5 mm. (*i. e.* the height of the mercury column=10 mm.), and the order of expansion of the different parts observed. It was as follows :

(1) Left apex.

(2) Right apex.

(3) Vertical strip along costal angles behind.

(4) Extension downwards over both back and front, most extensive on the right side, a few isolated patches refusing to expand.

(5) Slight bubbling below front of root of left lung.
(6) „ above „ „
(7) „ below „ right lung.
(8) „ to outer side of „ „

One patch of subpleural emphysema the size of a hempseed on the inner surface of the right upper lobe.

(Up to this time the rise of the mercury column did not exceed 10 mm [*i. e.* the height of the whole column=20 mm.]).

Bubbling continued all round the root of the right lung, especially below, the spot of emphysema increased, and one or two smaller ones formed near it.

Pressure was now increased to a rise of 15 mm. (*i. e.* height of whole column=30 mm.).

A large patch of emphysema formed at once on the inner side of each upper lobe, as large as half a marble.

(N.B.—These patches spread when the pressure only causes a rise of 5 mm., *i. e.* under a column of 10 mm).

One or two very small patches of emphysema were seen at the sides of the lungs, but none in front.

A large patch formed on the under surface of the right upper lobe, also in the fissures of the right lung.

The left lower lobe was then treated separately, the cannula was thrust into the lung substance and secured, the lung was then inflated. Immediately, a large bubble rose on the surface, disappearing as soon as pressure was relaxed, and reappearing instantly when it was resumed. It was found impossible to burst this even when 100 mm. pressure (*i. e.* a rise of 50 mm.) was steadily and repeatedly produced.

The bronchi being tied, it was found impossible to burst them under the greatest expiratory force available (=a rise of a 50 mm., or a column of a height=100 mm.).

*Remarks.*—The object was to see the behaviour of the lungs themselves uninfluenced by the thorax, (1) with regard to the order of expansion of different parts, (2) with regard to the comparative tenacity of different parts of the lungs, (3) of the pulmonary pleura, (4) of the bronchi.

(1) The order of expansion is given above. It is curious that the vertical strip along the costal angles, which is one of the last places to be expanded when the lungs are within the thorax, should have been one of the first to expand here.

(2) The weakest part of the lung was seen to be about its root, which gave way (subpleural emphysema) under the pressure of a column = 20 mm.

(3) The strength of the pulmonary pleura was seen to vary immensely; here it could not be ruptured, in former experiments the pleura was ruptured by far less pressure.

(4) The bronchi could not be ruptured by the strongest expiratory effort.

Emphysema, once formed, was seen to spread at a comparatively small pressure (column = 10 mm.).

EXPERIMENT 9.—November 8th, 1882. Fœtus female, born November 6—7 (midnight) and lived eight hours. Artificial respiration had been performed.

Experiment November 8th, 10 p.m. Thorax opened, but lungs not removed. No pneumothorax; a few small subpleural ecchymoses, lungs partly expanded.

Tracheotomy, cannula tied in trachea. Inflation (to a rise=10 mm., or a column=20 mm.) caused no emphysema.

The pressure was increased to a rise of 15 mm. (or a column = 30 mm.). Slight scattered interlobular subpleural emphysema followed, most marked in the left lung.

On repeating inflation under the same pressure, emphysema spreads, especially in the fissures.

The pressure was increased to a rise of 20 mm. (or a column = 40 mm.), and a bubble of subpleural emphysema on the inner side of the right middle lobe gave way.

After several repetitions, a large bubble was seen to occupy the posterior mediastinum, extending below and in front of the root of the left lung, and gradually extending upwards thence into the anterior mediastinum between the thymus and pericardium.

Although pressure was repeatedly increased to a rise of 25 mm. (or a column = 50 mm.) and although the posterior mediastinum was full of air as far as the diaphragm, no air rose into the neck.

*Remarks.*—The object was to observe the behaviour of the lungs *in sitû*, but with the thorax opened.

(1) No emphysema took place till a rise of 15 mm. (or a column = 30 mm.), *i. e.* the lung substance gave way at this pressure.

(2) The first emphysema was between the lobules at the surface of the lung.

(3) Emphysema appeared very early between the lobes.

(4) The pleura gave way at a rise of 20 mm. (or a column = 40 mm.)

(5) The air did not rise into the neck.

EXPERIMENT 10.—November 9th, 1882. Male, seven months child, born midnight November 6th—7th, died 12.30 p.m. November 8th (thirty-six hours).

Thorax opened. Tracheotomy, cannula in trachea.

On inflating the lungs, a leak is seen in the inner edge of the left upper lobe (the lung had probably been pricked in opening the chest). The leak cannot be stopped.

Experiment failed.

EXPERIMENT 11.—December 29th, 1882. Full-time stillborn male child, of somewhat doubtful freshness (date of birth unknown).

Thorax opened, cannula tied into trachea.

Lungs entirely airless, very watery.

Slight expiration inflates patches of the lungs, especially on the posterior surface.

Additional inflation produces increased expansion of the same parts, least behind.

Eventually the lungs became almost entirely expanded.

The pressure was increased to a rise of 10 mm. (or a column = 20 mm.). No emphysema.

The pressure was increased to a rise of 15 mm. (or a column = 30 mm.). Considerable emphysema of the front

of the root of the right lung occurred, extending into the fissure in front between the right upper and middle lobes, and also (but less) emphysema of the anterior and inner aspects of the upper and middle lobes. It began most markedly between the lobules. A bubble burst on the inner and anterior surface of the right upper and middle lobes.

Numerous leaks having occurred through the pleura the root of the right lung was tied.

Pressure producing a rise of 10 to 15 mm. (or a column = 20 to 30 mm.) produced well-marked emphysema of the front of the root of the left lung, extending in the anterior mediastinum and ½ inch along the course of the left phrenic nerve.

On repeating this, the inner surface of the upper lobe became emphysematous, and a bubble in that situation burst. Emphysema extended into the fissure between the upper and middle lobes.

The pressure was increased to a rise of 20 mm. (or a column = 40 mm.), and produced much escape of air from subpleural bubbles and extension of the emphysema from the root of the lung downwards into the posterior mediastinum.

The *bronchi were then tied,* and the greatest possible expiratory force exerted, causing a rise of 75 mm. (or a column = 150 mm.).

*No escape of air took place.*

*Remarks.*—(1) Emphysema (= rupture of the lung) took place at a pressure = a column 30 mm. high.

(2) The pleura eventually gave way at this pressure.

(3) The front of the root of each lung was the first place to give way.

(4) The spaces between the lobules and lobes (*i. e.* the interlobular spaces and fissures) were weak places.

(5) The bronchi and trachea could not be ruptured by the greatest expiratory effort.

EXPERIMENT 12.—December 30th, 1882. Large full-

time female child, stillborn (placenta prævia), about twenty-four hours.

Thorax opened; lungs unexpanded and very sodden.

Tracheotomy, cannula tied into trachea. Inflation was begun at a rise of 10 mm. (or a column = 20 mm.), and produced scattered inflated patches on the anterior and internal edges of all the lobes of both lungs, most marked inferiorly.

Second inflation at the same pressure produced an increase of the same, plus slight subpleural emphysema of the left base posteriorly.

The third inflation at the same pressure fully inflated the lungs and slightly increased the emphysema.

The pressure was increased to a rise of 15 mm. (or a column = 30 mm.), and produced a leakage of air from the left base.

The left base, seeming to be unusually frail, was tied off, and the experiment resumed.

On repeating the inflation at the same pressure a large patch of emphysema occupied the tip of the lingula. A large bubble formed in front of the root of the left lung, and smaller ones on the inner surface of the left upper lobe, mostly interlobular. The emphysema, still mostly interlobular, extended over the left lung.

The left lung was next tied at the root, and the experiment continued with the right lung.

Inflation was continued with a rise of 15 mm. (or a column = 30 mm.), which produced interlobular emphysema of the right base. Large bubbles occupied the fissures between the middle and lower lobes.

The pressure was increased to a rise of 20 mm. (or a column = 40 mm.); the emphysema extended over the front of the middle lobe, and a bubble burst, probably in one of the fissures.

The root of the right lung was tied and the strongest expiratory force exerted. No leak was produced at a rise of 80 mm. (or a column = 160 mm.).

*Remarks.*—The order of inflation was unlike the

former experiments, perhaps in consequence of the sodden state of the lungs.

(1) The left lung gave way (emphysema) under a column 20 mm. high, the right under a column 30 mm. high.

(2) The left pleura gave way under a column 30 mm. high, the right under a column 40 mm. high.

(3) The spaces between the lobules and lobes (interlobular spaces and fissures) were weak places.

(4) The bronchi and trachea could not be burst by the greatest expiratory effort.

EXPERIMENT 13.—January 1st, 1883. Full-time still-born child (craniotomy), born twenty-four hours previously.

Thorax not opened. Tracheotomy, cannula tied into trachea and attached to mercury manometer as before.

The lungs were inflated at a rise not exceeding 10 mm. (or a column 20 mm. high), then the thorax was compressed (the escape of air being prevented), causing an increased rise of 30 mm. (or a column 60 mm. high).

This was several times repeated, to imitate as far as possible the effect of bearing down.

A projection is seen above each clavicle, apparently from the apices of the lungs.

After several repetitions the left side of the neck was seen to be full, and on pouring water on the tracheotomy incision bubbles were seen to escape from the left side of the wound.

The initial pressure was now increased, so as to produce a rise of 15 mm. (or a column = 30 mm.), and then a rise of 20 mm. (or a column = 40 mm.).

*Autopsy.*—Considerable emphysema was found round the root of left lung, extending into the fissures and posterior mediastinum. Interlobular emphysema of whole of left lung, air escaping from several places. Slight diffused subpleural and interlobular emphysema of right lung, especially in front of its root.

No emphysema of neck.

The bronchi being tied, the greatest possible expiratory efforts, producing a rise of 80 mm. (or a column = 160 mm.), produced no escape of air.

*Remarks.*—(1) The dissection threw doubt on the escape of air from the neck, at least no air was found along the great vessels on dissection.

(2) The emphysema was seen to occupy the interlobular spaces, fissures, and the front of the root of the right lung.

(3) The bronchi and trachea could not be burst by the greatest expiratory effort.

EXPERIMENT 14.—February 22nd, 1883. Full-time still-born male child (face presentation), born February 19th.

Experiment February 22nd, noon. Thorax not opened. Tracheotomy, cannula tied into trachea.

The lungs were then inflated by intermittent inflations, the pressure causing a rise not exceeding 15 mm. (or a column = 30 mm. high), and these inflations were continued half an hour.

*Autopsy.*—Lungs completely inflated, no emphysema. Inflation was then continued at same pressure; no further result.

Pressure was then increased so as to produce a rise of 20 mm. (or a column = 40 mm.), producing a small spot of *subpleural emphysema* on the anterior surface of the internal inferior angle of the right middle lobe, and in several *interlobular* spaces on the inner surface of the right upper and middle lobes.

On repeating the inflation, the emphysema spread and extended in front of the root of the right lung, from which air then escaped.

The emphysema extended to the left lung and in front of its root, markedly between the lobules and in the fissures.

The emphysema then extended slightly into the right side of the *anterior mediastinum*, above and in front (from the front of the root of the right lung), and into the left side of the posterior mediastinum behind and below.

The emphysema then occupied all the fissures of both lungs.

The right side of the posterior mediastinum then became emphysematous.

Both lungs eventually showed great emphysema behind all pleural reflections.

*Remarks.*—The lungs were unusually air-tight; it was not till a rise of 20 mm. (or a column of 40 mm.) was produced that any emphysema appeared.

(1) The lung tissue gave way at a rise of 20 mm. (or a column of 40 mm.).

(2) The pleura gave way at the same pressure when prolonged.

(3) The pleural reflections and the interlobular spaces were the weakest. (These include the fissures and the reflection of the pleura over the root of the lung to the anterior mediastinum.)

EXPERIMENT 15.—February 27th, 1883. Male stillborn child (premature labour at end of eighth month), born February 26th, 2 p.m.

Experiment February 27th, noon.

Tracheotomy, cannula tied into trachea. Lungs were inflated at a pressure causing a rise not exceeding 15 mm. (or a column = 30 mm.). This was repeated intermittently for fifteen minutes, when the pressure was increased to a rise of 20 mm. (or a column = 40 mm.).

No signs of emphysema of the neck.

*Autopsy.*—No emphysema except a small spot on the inferior edge of the front of the right middle lobe (? produced in opening thorax), from which air escapes. On holding this, the emphysema spreads widely over all surfaces of the right lung. The right lung was then tied at its root. The left lung sustained a pressure causing a rise of 20 mm. (or a column = 40 mm.). On increasing the pressure to a rise of 40 mm. (or a column = 80 mm.), diffuse subpleural emphysema formed over posterior and inferior surface of back of left base.

*Remarks.*—The lung tissue only yielded to a pressure equal to a column of mercury 80 mm. high.

EXPERIMENT 16.—March 17th, 1883. Stillborn male child, born evening of March 15th.

Experiment at 11 a.m., March 17th. Thorax not opened. Tracheotomy, cannula tied into trachea.

The lungs were inflated intermittently, the pressure being gradually increased from a rise of 15 mm. (or a column = 30 mm.) to a rise of 40 mm. (or a column = 80 mm.), at which pressure the column sank as if from a leak.

*Autopsy.*—Air in both pleuræ, a good deal of fluid; lungs very œdematous. One or two very small patches of subpleural emphysema on both lungs. In front of the root of the right lung is a large patch extending into the anterior mediastinum in front, into the posterior mediastinum (which is greatly distended) behind, and into the fissure between the right middle and lower lobes.

On the left side the root of the lung is simply surrounded with emphysema, raising the reflection of the visceral and parietal pleura along the inner edge of the lung in its whole length, thus filling the posterior and left half of the anterior mediastinum. From the front of the root of the right lung bubbles extend to the phrenic nerve and along its course to the diaphragm, where there is a large collection of bubbles. No collection of bubbles is seen on dissection on either side of the neck, but they can be pressed from the anterior mediastinum upwards and escape alongside of the great vessels on the left side, passing behind the left innominate vein.

*Summary.*—Rupture of both lungs about their root at a pressure of 80 mm. of mercury; escape of air into both mediastina, on the left side following the phrenic nerve to the diaphragm. Pneumothorax (double), probably from escape of air from a rupture of the mediastinum.

*Remarks.*—The weak parts were the reflections of the pleura as before, especially the large ones in front of the root of the lungs and in the fissures.

Although air was not actually found in the neck, a free communication between the neck and the anterior mediastinum was demonstrated. The passage of air along the phrenic nerve to the diaphragm has already been noted in other cases and also in the experiments on artificial respiration ('Med-Chir. Trans.,' vol. lxv, 1882, pp. 77 and 80).

EXPERIMENT 17.—March 20th, 1883. Full-time stillborn male child (second of twins) born March 19th, 8 a.m.

Experiment March 20th, noon. Thorax not opened. Tracheotomy, cannula tied into trachea.

Lungs were inflated at a pressure producing a rise not exceeding 15 mm. (or column = 30 mm.)

Pressure was gradually increased to a rise not exceeding 20 mm. (or column = 40 mm.), giving twenty inflations at each rise of 5 mm. (or increase of column = 10 mm.).

On reaching this pressure, the *left side of the neck became distended, and air escaped from the left side of the incision.*

*Autopsy.*—No pneumothorax. The lungs on being inflated after opening the thorax are found perfectly air-tight.

*Left side.*—Air extends along the large vessels of the neck on the left side, and can be traced behind the left innominate vein to the anterior mediastinum, where a large collection is continuous with one in front of the root of the left lung and extending into the fissures of the lung. Air distends the posterior mediastinum and runs beneath the whole reflection of the visceral into the parietal pleura, along the inner edge of the left lung, and from thence along the diaphragm to the termination of the phrenic nerve; a few scattered spots of subpleural emphysema, especially on the "lingula."

*Right side.*—No emphysema of neck. Emphysema in front of root of lung and upwards a little way along phrenic nerve towards neck (N.B.—The middle lobe was not separated from the upper) and along the inner side of

the middle lobe following an intralobar fissure; also (as on left side) in posterior mediastinum, extending forwards to the ending of the phrenic nerve in the diaphragm, along the reflection of the pleura over the adjacent sides of the anterior mediastinum, diaphragm, and pericardium; also upwards along the phrenic nerves.

No other emphysema.

*Remarks.*—Emphysema of the left side of the neck was produced without pneumothorax. The air as usual had escaped from the front of the left lung near its root, into the mediastinum and along the great vessels to the neck. The fissures were also occupied by emphysema.

The pleura (though not the lung) withstood a pressure producing a rise = 20 mm. (or a column = 40 mm.).

*Consideration of the experiments.*—The production of emphysema of the neck was effected in the very first experiment, but it was not till Exp. 7 that it was produced without pneumothorax (left side). This result was also achieved in the last experiment, No. 17 (left side).

The difficulty was to find the necessary pressure.

The question of the significance of the cases in which pneumothorax was produced will be discussed hereafter.

The *route selected by the air* was exactly the same as in emphysema of the mediastinum after tracheotomy (after entering the mediastinum), but in a contrary direction.

The *source of the air* was rupture of the lung tissue producing interlobular emphysema near the anterior aspect of the root of the lung.

The question *why this spot is especially prone to rupture* was capable of two answers: (1) it might be due to inherent weakness in this part of the lung, (2) it might be due to the relation between the lung and the thoracic box.

This question was investigated by experiments on the lungs with the chest open, or after their removal from the chest. The results obtained showed that in lungs removed from the pressure of the chest wall, the weakest parts are the spaces between the lobules and lobes.

This is easily intelligible when it is remembered that the pleura invests the lung with hardly any adhesion to its surface, and that it can be stripped up with almost inappreciable force by air beneath it. The strength of the pleura is simply that of an independent investing elastic bag. If the air escapes from a lobule it finds no resistance from the pleura except on the surface of the lung, and may easily lie between the lobules without any pressure from the pleura.

With regard to the fissures of the lung, the same is true; the pleura is here, so to speak, slack and offers little or no resistance to air once escaped from the air-cells.

But experiments showed that the root of the lung was a specially weak spot with the thorax closed, though not with the thorax open. In other words, when the thorax was open it merely shared the weakness common to all the pleural reflections.

This point requires some discussion. What is the physical condition of the lung during inflation within the closed thoracic walls? The thorax is distended, the diaphragm depressed, the sternum, clavicles, and ribs elevated.

Is the thorax or the lung the more distensible? To this it must be answered that the lungs can easily be ruptured within the thorax.

But it nevertheless cannot be doubted that, up to this point, the thorax supports its contents.

Does it do so equally in all directions? Below, we have the unbroken plane of the diaphragm; behind, in front, and at the outer side we have the ribs and muscles. But at the inner side we have the compressible mediastinum, whose easy penetrability was proved in the experiments already alluded to dealing with the subject of mediastinal emphysema after tracheotomy. This side then seems to be the direction of least resistance.

When once in the mediastinum, the air is already within the track of easy penetrability or of slight resistance, leading into the neck, and which probably owes

this quality partly to the fact that the upper aperture of the thorax is the weakest spot in the thoracic box, not being directly defended by muscles, which pass obliquely from the neck to the upper ribs, clavicles, and sternum. It must also be remembered that this upper aperture of the thorax is enlarged on inspiration.

The above reasons show that the mediastinum may be considered to be within the area of diminished resistance.

What happens in a bearing-down effort? First a deep inspiration, which (among other things) raises the upper ribs and clavicles, and increases the size of the upper aperture of the thorax. Next the glottis is closed. Lastly the whole of the expiratory muscles, essential and accessory, put forth their strength.

The least resistance is offered to pressure at the upper aperture of the thorax.

Thus then, on the *anterior surface of the root of the lung is the pleural reflection least supported externally.*

We have now to consider the question of the significance of the occurrence of pneumothorax in some of the experiments.

Is the pneumothorax in these cases a link in the chain ending in emphysema of the neck?

A very little consideration will suffice to put such an idea aside.

Let us consider the course of the air on this hypothesis. It escapes from the air-vesicles beneath the pleura; bursts through the pulmonary pleura; must then distend the pleural cavity; then bursts through the parietal pleura, and so gets beneath the deep cervical fascia.

First of all, the autopsies entirely contradict such an assertion; they show the course of the air to be different.

Secondly, the air beneath the pulmonary pleura finds practically no resistance in travelling beneath the pleura, but the pulmonary pleura is rather tougher than the lung. The experiments show the tenacity of both lung and pleura to vary very greatly in the fœtus (whatever they do in the adult), as the following table shows:

*Lowest force required to burst the lung :—*

| | | |
|---|---|---|
| Experiment No. 8 | . . | 20 mm. |
| „ „ 12 | . . | 20 „ |

*Lowest force required to burst the pleura :—*

| | | |
|---|---|---|
| Experiment No. 11 | . . | 30 mm. |
| „ „ 12 | . . | 30 „ |

*Highest force required to burst the lung :—*

| | | |
|---|---|---|
| Experiment No. 15 | . . | 80 mm. |

*Highest force required to burst the pleura :—*

| | | |
|---|---|---|
| Experiment No. 8 (N.B., locally) | | 100 mm. |
| „ „ 9 | . . | 40 „ |
| „ „ 14 | . . | 40 „ |

An attempt was made (Exp. 3) to test the tenacity of the parietal pleura, but it was unsuccessful. It must, however, be remembered that the tenacity of a membrane like the pleura must be very different when raised from the subjacent structures (pulmonary pleura), and when subjected to force, which only presses it the more firmly on its supports. It can easily be imagined that a film of collodion might add great strength to a membrane if force was applied in such a direction as to press the film against the membrane, while its strength would probably be very small if it had to resist a force from below, that is, raising it from its supporting membrane.

We therefore conclude that pneumothorax, when it occurred, had nothing to do with the production of emphysema of the neck.

We have avoided speaking of the clinical phenomena which form the actual subject of our consideration, but it must not be forgotten that pneumothorax never occurs either with emphysema of the neck during labour, or as the result of expiratory efforts.

We have not hitherto spoken of the theories that regard

emphysema of the neck as due to rupture of the trachea or of the bronchi.

This may be dismissed in a word: *The strongest expiratory effort failed to burst the trachea or bronchi of a fœtus.*

The experiment was repeatedly tried, and amounts to a "reductio ad absurdum" of the hypothesis.

It cannot be alleged that in the cases in which this accident occurs during labour the patients have had disease or fistula of their bronchi or trachea, for the patients may in all cases be, and are nearly always, expressly described as perfectly healthy, and moreover they suffer no ill-effects.

To the question whether a lung can be ruptured with impunity we must answer in the affirmative; if the air is all absorbed within a week from the subcutaneous cellular tissue, why not from beneath the pleura?

Again, the collection beneath the pleura must be small or it would give physical signs, or at least marked symptoms, which, however, are always absent.

What actually happens is probably as follows: During a violent expiratory effort an air-cell near the front of the root of the lung gives way, and the air lies beneath the pulmonary pleura. With the next effort this becomes larger, and part of it moves in the direction of least resistance, namely, towards the mediastinum, next time towards the neck, and so on, until a bubble emerges beneath the deep cervical fascia. A channel will thus be formed along which bubbles will pass as quickly almost as they escape from the lung. They may form a large collection in the neck, beneath the deep cervical fascia, where the pressure is small; eventually they may find their way into the superficial fascia, and so all over the body.

The gelatine injection (Exp. No. 4) gave us the interesting fact that a lung which is quite air-tight is quite permeable for fluids. I had previously proved this as regards water, and it is seen to be true also of a colloid mass. This fact has various and important bearings, which this is not the place to enlarge upon. It may,

however, be remarked that the air-cells are lined with an epithelium not much removed from an endothelium, such as that which lines serous cavities which are lymph-sacs and highly permeable to fluids.

The experiments are thus seen to illustrate the clinical facts of emphysema of the neck during labour, and during violent expiratory efforts.

The emphysema is essentially *expiratory* in its nature, and due to a cause entirely opposite to that which is answerable for emphysema of the mediastinum after tracheotomy. In saying this it is conceivably possible that expiratory emphysema might occur after tracheotomy, though the conditions under which the operation is performed are well known to obstruct inspiration rather than expiration. In this case the operation would have nothing to do with the emphysema. Emphysema of the lung may occur from over-distension of a part due to obstruction in other parts. This is a well-known clinical fact, and was also illustrated in the experiments quoted above ('Med.-Chir. Trans.,' vol. lxv, 1882, p. 78, Exp. 19 (L.). In such a case the air which had escaped beneath the pulmonary pleura might conceivably be forced into the mediastinum. But the burden of proof in any case of tracheotomy rests with the observer who asserts that the emphysema which he finds, is due to expiratory rather than inspiratory causes. The mediastinal emphysema of tracheotomy is *inspiratory*; the mediastinal emphysema of violent expiratory efforts is *expiratory*. Mediastinal emphysema without subpleural emphysema cannot be expiratory. Subpleural emphysema without mediastinal emphysema may be inspiratory as well as expiratory. Subpleural, together with mediastinal, emphysema is probably altogether expiratory, but the former may be due to inspiratory over-distension of part of the lung with obstruction elsewhere; the latter may conceivably be due to the expiratory forcing of the air thus escaped into the mediastinum. We are now only speaking of cases of tracheotomy in which the derivation of the air from a cervical

wound is a possibility, and the source of the air is therefore so far debateable.

But while allowing the above possibilities, we wish to repeat that the emphysema of tracheotomy occurs under conditions which obstruct inspiration, and is therefore essentially *inspiratory*, the air being derived from the cervical wound.

The following conclusions are offered :

1. The cause of emphysema of the neck during labour is rupture of the lung tissue, the air escaping near the root of the lung, passing beneath the pulmonary pleura into the anterior mediastinum, and so beneath the deep cervical fascia into the neck. The route thus marked is the same by which air sometimes passes into the anterior mediastinum after tracheotomy (see 'Med.-Chir. Trans.,' vol. lxv, 1882, p. 75, *et seq.*, and p. 85).

2. The weakest parts of the lung are opposite the pleural reflections (that is the fissures) and the interlobular spaces. The anterior surface of the root of the lung is the weakest spot while the lungs are within the thorax, being that pleural reflection lying within the comparatively unsupported area near the upper aperture of the thorax.

3. Pneumothorax, when it occurred during experiment, had nothing to do with the production of emphysema of the neck, and in two experiments was not associated with this emphysema, which thus exactly imitated that occurring during labour.

4. The healthy bronchi and trachea are able to resist the greatest possible expiratory efforts.

5. The lungs and pleuræ when quite air-tight are freely permeable to fluids.

6. The usual rules of practice to restrain bearing down and accelerate labour after the production of emphysema of the neck are sound.

7. The accident would seem to be noted in about 1 case in 2000, but it is not improbable that slight cases are overlooked.

8. The air emerges from the thorax along the great vessels, but may not become superficial till it has travelled higher up.

9. The emphysema of the lower part of the trunk, usually connected with rupture of the uterus, belongs to quite a different category and is generally associated with a fatal result.

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[For discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 285.]

# A SUCCESSFUL CASE

OF

# LUMBAR NEPHRECTOMY FOR RENAL CALCULUS.

BY

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(Received May 21st—Read November 25th, 1884.)

Reuben W—, æt. 35, a labourer, was readmitted into the Middlesex Hospital on the 13th of October, 1883, for the purpose of undergoing an operation for the relief of severe suffering due to renal calculus.

He had been a patient in the hospital on three previous occasions, and an exploratory incision had been made, but had not led to the detection of the calculus.

In May, 1882, he first came under the notice of Dr. Douglas Powell, whose notes of the case were kindly placed at my disposal. The patient at that time was described as a dark-haired, stoutly-built man, complaining of pain in his right side; the pain was not constant, and varied both in severity and position. It was sometimes felt in the epigastrium, at others in the right testicle, thigh, and loin, and shooting down to the bladder. Occasionally it was so severe as to completely "double him

up." Palpation of the right iliac region gave pain, but steady pressure often gave relief. The urine was 1035, acid, and contained a little blood. His symptoms commenced at the end of 1881 (five months before admission) with cutting pains in the pit of the stomach and right loin, disturbing his rest throughout the night, but more especially towards the morning. He had gradually become worse.

Eleven years ago he had scarlet fever; eight years ago he was disabled for three months by a blow on his head. He is prone to catch cold, and attributes the onset of his illness to lying in a damp barn. He remained under Dr. Powell's observation for a month, during which time his urine was frequently examined. On the average he voided 40 ounces a day, of specific gravity 1025—1035, acid in reaction, and containing a trace of albumen.

In November, 1882, he was admitted under me, and it was at this time that I explored his kidney through an incision in the loin, both with my fingers and the acupuncture needle. This operation seemed to give him temporary relief, but soon after leaving the hospital in December, 1882, his pains returned worse than ever, and he passed a large quantity of blood with his urine. He also passed two small stones about the size of pins' heads, but no relief followed.

In May, 1883, he was readmitted, and remained from the 1st to the 31st of this month. At this time pressure over the loin caused pain along the course of the right ureter, and in the right iliac fossa. The urine was 1025, acid, and contained a little blood and pus, and crystals of phosphates. There was nearly always a little pus in it, but blood was less constant and always very limited in amount. Sometimes the urine was perfectly clear, of sp. gr. 1005—1010; at others thick and high coloured, but it was always acid. When clear a much larger quantity was voided.

Jolting movements, such as riding in a cab or railway carriage, and muscular movements, did not increase his

pain. Paroxysms of very severe pain came on whilst he lay quite quiet, and he was frequently awoke out of sleep by them. It seemed as if any increase of intra-renal tension, or of pressure of the bowel upon the kidney, excited these paroxysms. There was a certain periodicity about their occurrence; 12 midday and 1 a.m. being the times of most severe suffering.

Another exploratory incision, to be followed by nephrectomy if no calculus could be detected, was proposed, but the patient desired to return home first.

On October 13th, 1883, he came back to me worse than ever; a thin, haggard, worn-out looking man, with all the old symptoms aggravated, and having paroxysms of excruciating pain at all hours and under all circumstances. The pain was still all on the right side. His urine had the same characters as before. He had passed another calculus a little larger than a swan shot; its passage caused great suffering, but its escape was not followed by relief. On October 23rd he was in great pain nearly the whole day, and a small calculus, black and facetted, and of the size of a pin's head, was found in the urine.

On October 24th the patient was put under an anæsthetic, and a transverse incision about three inches in length was made in the right loin half an inch above the old cicatrix. Having thoroughly explored the kidney on both surfaces and compressed it all over between my fingers and thumb, and having also punctured it, I failed to detect any stone. The whole kidney was unusually hard, but no one part was harder or more resistant than the rest.

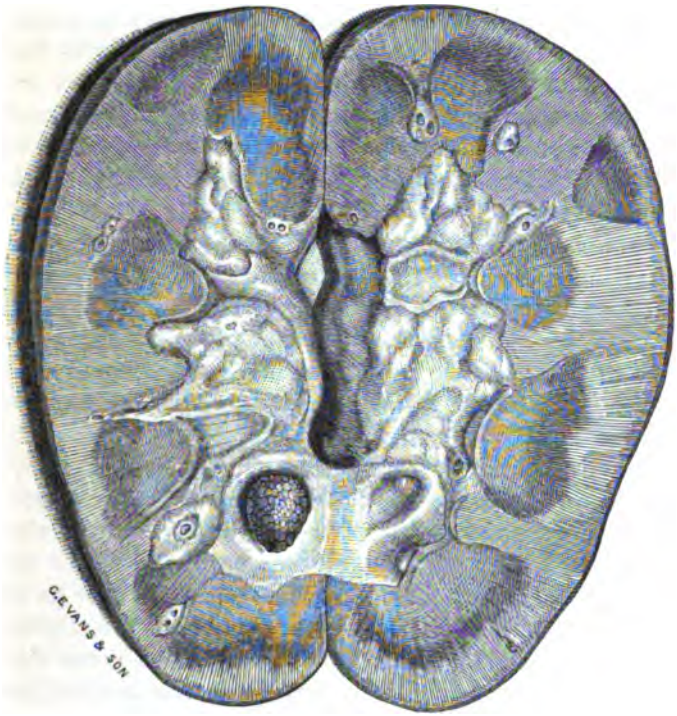
With the assistance of Mr. Gould and the house surgeon, Mr. Thornton, I removed the kidney through the loin wound in the following manner: After completing the separation of the organ from its surroundings I made a vertical incision downwards from the transverse wound, and one inch in front of its hinder end. This was done by cutting from within outwards with a straight probe-pointed knife introduced upon the left index finger. This second incision

much facilitated the application of the ligatures. In an aneurism needle specially provided with a long handle, a long loop of twisted silk was passed, as I believe, between the ureter and blood-vessels. The silk loop was divided and the vessels tied with one half and the ureter with the other. With the fingers of the left hand I then forcibly dragged upwards the lower ribs, whilst with the right hand I drew the kidney on to the surface of the body. Another ligature was now applied so as to include the whole pedicle. This was tied firmly. The kidney was next cut away with a pair of broad-ended scissors. As the renal pelvis was divided a rounded rough calculus dropped out of the kidney into the wound. The ligatures were all cut off short. During the process of dragging the kidney from its cushion of fat its surface was broken, and for a few moments there was smart hæmorrhage from the laceration. This led me to be as rapid as possible in completing the removal and applying the ligatures, without waiting to attempt to stay the bleeding by pressure. After the kidney was removed a medium-sized vessel in the depth of the wound gave a little trouble, but was at length secured by a pair of Wells' forceps. These forceps were left in for twenty-four hours, and served as an excellent means of drainage in addition to the drain-tube. The edges of the wound were sutured together, a drainage tube was inserted, and terebene oil on lint, and a pad of absorbent cotton wool were retained on the loin by means of a gauze bandage.

It is needless to report in detail the after-progress of the case, as nothing occurred to interrupt recovery.

On the fifth day (October 29th) he complained of pain at the commencement of micturition and continued to do so till November 7th. On the sixth day the stitches were removed. On the seventh day drainage was discontinued. On the tenth day sleeping draughts were no longer needed. On the twelfth day the bowels acted for the first time, and in response to an enema.

On November 22nd the patient sat up for the first time.




Calculus as seen *in situ* in the kidney.

November 29th.—Two decalcified bone drainage-tubes were inserted into long narrow sinuses; the rest of the wound had entirely healed.

December 11th.—He left the hospital in good health, but with one of the sinuses still unhealed.

The examination of the kidney after its removal disclosed a rounded depression in one of the calyces towards the lower end of the organ. This depression was lined with a thin cyst-like membrane and was spotted over with several minute ecchymoses. The calculus exactly fitted into it, and had doubtless there lodged till it was displaced by the manipulations necessary for the excision of the kidney.

Dr. Coupland kindly undertook the microscopical examination and found the kidney structure to be quite healthy. The kidney was of normal size and of great hardness. The calculus as it lay in its depression in the kidney was so thickly surrounded by the renal substance that it could not be detected *by pressing the kidney with the fingers as it rested on a table.*

On July 28th, 1884, I heard from Dr. Frederick Pearse, of Haslemere, to this effect: "W— is hard at work 'charcoal burning;' the sinus still discharges a watery sero-purulent fluid enough to soak through four or five pieces of thin rag in twenty-four hours. He feels as well as ever he did in his life, and is able to do his work without the slightest inconvenience. Three or four threads have come away through the sinus since his return from the hospital, and he says there is another working out at the present time."

On November 12th, 1884, Dr. Pearse wrote again: "I saw W— a week ago. He is in excellent health and at hard work (charcoal burning). The wound is not quite closed yet; there is still a little watery sero-purulent discharge from it."

*Remarks.*—This case illustrates the great difficulty which must occasionally be expected in determining the presence of a stone in the kidney by digital exploration and acupuncture. The general symptoms pointed con-

clusively to renal calculus; but it was an open question whether the disease was in the form of nephrolithiasis, or of one or more distinct calculi of larger size.

The case teaches us not to conclude that a calculus, even of moderate size, is not present, still less that no calculus at all is there, because we cannot discover it either by probing with a needle or by compressing the kidney between the fingers and thumb. It teaches also that the abdominal incision, no more than the lumbar incision, can lead to the detection of a stone when it is thickly surrounded by renal tissue. The position of the stone could not be made out either by the eye or the fingers, when the kidney was out of the body and resting upon a table. It certainly could not have been detected whilst the kidney remained in its fatty cushion in the loin. The prolonged irritation of a calculus in the kidney appears to have had a hardening effect upon the kidney tissue, and in this way to have assisted in completely masking the presence of the calculus.

Two questions arise with respect to the operative treatment of similar cases of calculus in the kidney.

1. Before resorting to nephrectomy can any other step be taken with the view of ascertaining the whereabouts of a stone which has escaped detection by digital examination and needle probing?

2. Is the lumbar, or one of the anterior abdominal incisions, the best for nephro-lithotomy, and for nephrectomy for calculous kidney?

In answer to the first question I would urge that the renal substance should be incised from behind, so as to lay open each of the calyces one after the other until the stone is found. This of course is advised only for cases in which the symptoms are severe and point in a marked manner to calculus in one kidney. Our knowledge of wounds of the kidney gives us assurance that hæmorrhage from such an incision would be easily controlled, that the wound though large would readily cicatrize, and a good working kidney would remain for future service.

The risks of such an incision are much less than those of nephrectomy, and the patient would have the incalculable advantage of being left with two kidneys instead of one.

In answer to the second question I think the lumbar incision is to be preferred for nephrectomy for calculous kidneys, and that it is the only incision which ought to be employed for nephro-lithotomy.

The arguments which have been advanced in favour of the anterior or abdominal nephrectomy are: (*a*) when the peritoneal cavity is opened there is more room for the necessary manipulations; (*b*) that the surgeon can see all that he is doing, and can secure the renal vessels before commencing to enucleate the kidney; (*c*) that the existence of a second kidney, and its condition, can be ascertained, and the operation abandoned, if only a solitary kidney or a diseased second kidney be discovered; (*d*) that the end of the ureter can be brought out of the wound, and that thus the danger of suppuration along the tube can be avoided; (*e*) that in certain cases, such as hydronephrosis, pyonephrosis, and hydatid and simple cystic enlargements, the peritoneal method, by making evident the nature of the disease, has enabled the surgeon effectually to treat the case by incision and drainage, and thus to do away with the necessity of nephrectomy.

Of these reasons the first two have only an occasional importance, and need not be considered in calculous affections; whilst the others are for the most part purely theoretical, and may sometimes lead to pernicious results.

That an abdominal operation commenced with the intention of nephrectomy, may end advantageously by simply opening and draining the kidney is no more in favour of the incision through the peritoneum than of the lumbar method. In most cases in which such treatment is possible the cystic character of the disease can be diagnosed without any exploratory incision; the cyst can as readily be opened behind the peritoneum as through it; and the margins of the cyst are as easily stitched to the margins

of the wound in the loin as to those of an incision on the front of the abdomen.

I have thus dealt most satisfactorily through a lumbar incision with the kidney when enormously enlarged by hydronephrosis. The drainage afforded by the dependent position of the loin wound leaves nothing to be desired, and in this respect the lumbar method has a very great advantage over any anterior abdominal procedure.

For the same reason—the advantageous drainage—the divided ureter is best dealt with through the loin. In most cases, particularly of calculous disease, the ureter may safely be left to take care of itself. When fixed to an anterior wound there is an undeniable risk of intestinal obstruction; whereas in lumbar nephrectomy, if suppuration occur about the end of the ureter, either as the effect of its own diseased state or of the ligatures on the renal vessels, the pus can readily escape at the wound, instead of forming an abscess in the abdominal cavity.

Dr. Williston Wright,[1] in a case reported in the 'New York Medical Journal,' brought the ligatures out at the lower end of an incision along the outer edge of the rectus muscle (Langenbuch's incision); intestinal obstruction resulted, and an abscess which formed about the stump of the renal vessels and ureter failed to empty itself through the operation wound, but fortunately did so through the ureter and bladder.

It seems to me to be anatomically wrong to drag the ureter from the back of the abdomen in order to attach it to the front, thus stretching it across the peritoneal cavity. So that even if it should ever be proved the better practice to open the peritoneum in cases in which an operation could otherwise effect the same end without injuring that structure, I do not think that anything could be urged in favour of this treatment of the ureter.

Respecting the examination of the opposite kidney the argument seems plausible, but practically is almost useless. In the first place there are many cases in which the

[1] 'New York Med. Journ.,' Feb. 17, 1883.

nature of the disease, the character of the urine, and the general condition of the patient make clear the diagnosis as to the existence and soundness of the second kidney. In the second place, of what value is a digital examination of a kidney as it lies in the body? Who, holding the kidney enveloped in its capsule and fat in his hand in the post-mortem room, can do more than form an opinion as to its outline and size? Who can thus judge of its structure? Who can say that it is not fatty, granular, lardaceous, tubercular, cancerous, congested, inflamed, suppurating, or the subject of thrombosis? We know the kidney may be irreparably diseased without being enlarged or shrivelled, or without presenting any other character which is recognisable until it is deprived of its capsule or split into halves. So that in spite of a digital examination of the opposite kidney, and even partly because of it, the fatal mistake may be made of performing nephrectomy and leaving behind an organ seriously diseased and utterly insufficient.[1]

If it be true that there are cases in which the normal amount of urea is excreted by the hypertrophied areas of two kidneys, the other areas of which are diseased, the instances of this kind in which nephrectomy will be thought of are infinitely rare; and the pathological condition is not likely to be recognised by an intra-abdominal examination. Where there is a quantity of circumrenal fat it is just possible that even a shrivelled kidney could not be recognised as such. We know a calculus may be undetected.

When there is a large solid tumour, or a painful floating tumour (presumably a floating kidney) to be removed, and owing to some congenital deformity or deficiency, especially of the genital or urinary apparatus, a single kidney is suspected, abdominal nephrectomy should be performed. Therefore, without stating that

[1] A case of nephrectomy for scrofulous kidney which gave support to this statement was mentioned in my reply to the discussion on this paper (see 'Proceedings of Royal Medical and Chirurgical Society,' New Series, vol. i, p. 299).

peritoneal nephrectomy is never the better operation, I venture to assert that the lumbar method is the right one in calculous disease. It is so for these reasons: first, because up to the present time the mortality of the lumbar operation has been only half that of the peritoneal; secondly, the lumbar operation leaves the peritoneal cavity unopened; thirdly, the kidney is of a size to allow its easy removal through the loin, especially if the ilio-costal space be increased, as it ought to be during the operation, by forcibly dragging upwards with the left hand the lower ribs; and fourthly, the loin wound is the most favorable for drainage.

As to nephrolithotomy, I know of no argument which favours the abdominal incision, or the combined abdominal and lumbar incisions in that operation. All that can be ascertained by the anterior incisions, about the organ to be operated upon, can be discovered by the lumbar incision. The incision of the pelvis renalis, or secreting substance, in search of or for extracting a calculus, can only be safely performed behind the uninjured peritoneum; whereas, the condition of the opposite kidney would not deter one from the operation. On the contrary, if the second kidney is diseased it becomes more urgent not less so to extract the stone, and thereby to place the kidney operated upon in a better state for aiding its fellow, or performing the whole of the excretion.

As all mechanical measures which have been suggested for ascertaining the character of the urine secreted by the opposite kidney have proved to be either impracticable or unreliable, we must depend upon the general symptoms of each case, and upon the estimation of the daily excretion of urea.

In connection with this subject two facts are forced upon the attention by the present case. The first is that we must not infer that the kidneys are diseased because they do not excrete the average daily quantity of urea according to the standard usually given for a healthy adult.

Persons who have long been living an invalid life, feeding on an invalid diet, taking no exercise and therefore having little or no appetite, and who in consequence of such a life and of suffering and sleeplessness have lost flesh, may have very sound kidneys though they do not excrete more than 250 grains (*i. e.* not half the standard quantity) of urea in twenty-four hours. An adult under these circumstances with two healthy kidneys may not eliminate more than from ·8 to 1·8 per cent. of urea in from 25 to 35 ounces of urine.

Another remarkable fact is the rapidity with which the single kidney increased its power of eliminating urea, though it continued for a long time to discharge a smaller quantity of water than the two kidneys together had done. This is partly to be explained perhaps by the diet. The object was to diminish the nitrogenous matters and fluids as much as possible for the first week or two after the operation. Thus though nearly all the food was taken more or less in a liquid state, there was on the whole less fluid consumed than there would have been under ordinary circumstances. In the week immediately preceding the operation, the daily average quantity of urine passed by this patient was 35 ounces, and the average daily quantity of urea 248 grains.

Five hours after nephrectomy 2½ ounces of urine of sp. gr. 1022, and containing nearly 11 grains of urea were passed; and three hours later 1½ ounces of urine of sp. gr. 1040 and containing nearly 5½ grains of urea. During this time a little iced water and a little milk were the only things swallowed.

In the next five days the average daily quantity of urine was 28 ounces, and of urea 196⅓ grains. In the second twenty four hours after the operation the one kidney excreted exactly 196 grains, *i. e.* nearly four fifths of the daily average of the two kidneys just prior to the operation. This was a very sudden rise as compared with the first twenty-four hours after the operation; during this time 59½ grains of urea were eliminated in

addition to the 16½ grains of the evening of the day of operation, which together made a total of 76 grains in the first twenty-four hours.

The quantity of urine in the first twenty-four hours was only 11¾ ounces; in the next twenty-four hours it was 28 ounces.

Between the seventh and twelfth days inclusive the daily average of urea was 193 grains, and of urine 23½ ounces—or 55 grains of urea less, and 11½ ounces of urine less than had been excreted by the two kidneys just previous to the operation.

On and after the third day from the operation the diet consisted of chicken jelly, egg, tea, bread and butter, coffee, milk, and custard.

From the fourteenth day the urea increased considerably. It was very rarely below 200 grains, nearly always above 230 grains, often above 260 grains, occasionally above 320 grains. Even on the fifteenth day 296½ grains, and on the thirty-sixth day as much as 385 grains were excreted.

On the thirtieth day 45 ounces of urine, containing nearly 276 grains of urea were discharged, but with this single exception the daily quantity of urine did not increase until the sixth week. On the thirty-fifth day and onwards till the patient left the hospital the normal quantity, and occasionally even much more than the normal quantity of urine was passed.

I am indebted to Mr. Paul for the regular and careful examination of the urine over a very prolonged period. The results are of the nature of those derived from a physiological experiment, as the kidney removed was proved to be healthy, and the kidney remaining has proved itself so.

[For discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 291.]

*Postscript.*—On August 28th, 1885, Dr. Pearse wrote: "R. W— told me that a week ago a piece like string came from the wound, which quickly ceased to discharge, and is now healed up."

*Condition of urine before the operation.*

| Date. | Amount of urine in 24 hours. | Specific gravity and amount of urea in urine. | | Other characters of urine. | Diet. |
|---|---|---|---|---|---|
| 1882 | | Sp.gr. | Urea. | | |
| May 8th | ... | 1035 | ... | Acid; albumen; blood. | |
| „ 13th | ... | 1030 | ... | Clear; albumen $\frac{1}{8}$th. | |
| „ 25th | ... | 1025 | ... | Cloudy; alkaline; no albumen. | |
| „ 27th | ... | 1030 | ... | Acid; pale; no albumen. | |
| „ 28th | 40 oz. | | | | |
| „ 29th | 38 oz. | 1035 | ... | Small trace of albumen. | |
| „ 30th | 24 oz. | | | | |
| June 1st | ... | 1030 | ... | No albumen; phosphates. | |
| „ 2nd | 40 oz. | 1035 | ... | Slightly albuminous. | |
| „ 3rd | 60 oz. | | | | |
| „ 5th | 32 oz. | | | | |
| „ 8th | ... | 1030 | ... | Trace of albumen. | |
| „ 12th | ... | 1030 | ... | Albumen $\frac{1}{8}$th; no blood. | |
| 1883 | | | | | |
| May 8th | ... | 1025 | ... | Acid; slight trace of albumen; no blood. *Microscopically.* — Blood-cells; one or two pus-cells; phosphatic crystals. | |
| „ 14th | ... | 1020 | ... | Slightly alkaline; large trace of albumen; no blood; quantity of pus. | |
| „ 22nd | ... | ... | ... | Pale and clear; no albumen, blood, or pus. | |
| „ 26th | ... | 1010 | ... | Acid; some albumen; large quantity of blood; no pus. | |
| „ 28th | ... | 1025 | ... | High colour; albumen; no blood; some pus. | |
| „ 29th | 36 oz. | 1010 | ... | Acid; albumen; no blood or pus. | |
| „ 30th | 32 oz. | | | | |
| „ 31st | 40 oz. | | | | |

| | | | | | |
|---|---|---|---|---|---|
| Oct. 14th | ... | 1018 | ... | Acid; albumen $\frac{1}{10}$th. | |
| „ 18th | 42 oz. | ... | 294 grs., or 1·6 p.c. | | |
| „ 20th | 40 oz. | 1012 | 210 grs., or 1·2 p.c. | Acid; cloudy; trace of albumen. | |
| „ 21st | 22 oz. | 1022 | 173·25 grs., or 1·8 p.c. | Cloudy; albumen $\frac{1}{4}$th; a little pus; phosphates. | |
| „ 22nd | 38 oz. | 1022 | 315·875 grs., or 1·9 p.c. | Acid; trace of albumen. | |

*Condition of urine after the operation.*

| | | | | | |
|---|---|---|---|---|---|
| Oct. 24th, 7.15 p.m. (*Operation about 2 p.m.*) | 2½ oz. | 1022 | 10·9375 grs., or 1 p.c. | Acid; high coloured; cloudy; trace of blood; mucus; no pus. | |
| Oct. 24th, 10.15 p.m. | 1½ oz. | 1040 | 5·46875 grs., or 1 p.c. | High coloured and cloudy; scarcely perceptible trace of albumen; no blood or pus. | |
| Oct. 25th, 1.15 p.m. | 8 oz. | ... | 59·5 grs., or 1·7 p.c. | Acid; high coloured; clear; no albumen, blood, or pus. | Iced water and milk. |
| Oct. 26th | 28 oz. | 1028–32 | 196 grs., or 1·6 p.c. | Acid; very cloudy with lithates; no blood; slight deposit of mucus. | Arrowroot, egg, coffee and milk, lemon water, custard. |
| „ 27th | 36 oz. | 1031 | 315 grs., or 2 p.c. | Acid; no albumen. | Tea, egg, biscuit, chicken jelly, water, buttered toast, milk and coffee, custard. |
| „ 28th | 25½ oz. | 1031 | 176·75 grs., or 1·6 p.c. | High coloured; no albumen. | Oysters, sweet jelly, iced water, milk, tea and coffee, egg, chicken jelly. |
| „ 29th | 25¾ oz. | 1031 | 78·859375 grs., or ·7 p.c. | Acid; no albumen. | Ditto. |
| „ 30th | 25 oz. | 1036 | 215·3125 grs., or 1·9 p.c. | Acid; high coloured; no albumen; lithates; phosphates. | Ditto; pheasant and mashed potato. |
| „ 31st (*7th day*) | 18 oz. | 1032 | 126 grs., or 1·6 p.c. | Acid; no albumen; lithates. | Arrowroot, chicken jelly, water, sweet jelly, fish. |
| Nov. 1st | 23 oz. | 1026 | 201·25 grs., or 2 p.c. | Acid; high coloured; lithates. | Chicken, oysters, tea, toast and egg, water, arrowroot. |

24 hours

24 hours

| Date. | Amount of urine in 24 hours. | Specific gravity and amount of urea in urine. | | Other characters of urine. | Diet. |
|---|---|---|---|---|---|
| 1883 | | Sp. gr. | Urea. | | |
| Nov. 2nd | 36 oz. | 1027 | 283·5 grs., or 1·8 p.c. | Thick deposit of lithates. | Chicken, oysters, tea, toast and egg, water, arrowroot. |
| „ 3rd | 17 oz. | 1031 | 126·4375 grs., or 1·7 p.c. | Lithates. | Ditto. |
| „ 4th | 24 oz. | 1027 | 231 grs., or 2·2 p.c. | Lithates. | Ditto. |
| „ 5th | 24 oz. | 1027 | 189 grs., or 1·8 p.c. | Phosphates and lithates. | Ditto. |
| „ 6th | 24 oz. | 1030 | Undetermined | Acid. | Ditto. |
| „ 7th (*14th day*) | 26 oz. | 1032 | 284·372 grs., or 2·5 p.c. | Acid. | Fish, chicken jelly, eggs, milk, arrowroot, tea, oysters. |
| Nov. 8th | 31 oz. | 1032 | 296·25 grs., or 2·8 p.c. | Acid; lithates in abundance. | Ditto. |
| „ 9th | 24½ oz. | ... | 267·96872 grs., or 2·5 p.c. | Acid; phosphates. | Ditto. |
| „ 10th | 23½ oz. | 1034 | 246·75 grs., or 2·4 p.c. | ... | Ditto. |
| „ 11th | 28 oz. | 1036 | 245 grs., or 2 p.c. | Acid. | Ditto, with custard instead of fish. |
| „ 12th | 23½ oz. | 1036 | 213·609 grs., or 2·1 p.c. | Full of lithates. | Fish taken; otherwise the same. |
| „ 13th | 20 oz. | 1024 | 192·5 grs., or 2·2 p.c. | Acid; lithates. | The same, with six oysters. |
| „ 14th (*21st day*) | 28 oz. | 1037 | 261·625 grs., or 2·6 p.c. | Full of lithates. | Ditto. |
| Nov. 15th | 25 oz. | 1034 | 262 5 grs., or 2·4 p.c. | Alkaline; albumen; blood; no pus; quantity of mucus. | Ditto. |
| „ 16th | 26 oz. | 1030 | 250·25 grs., or 2·2 p.c. | Alkaline; cloudy; albumen $\frac{1}{10}$th; mucus; no blood or pus. | Tea, bread and butter, milk, milk pudding, lemon water, toast. |
| „ 17th | 32 oz. | 1022 | 238 grs., or 1·7 p.c. | Alkaline; cloudy; light colour; full of mucus; no albumen, blood, or pus. | Ditto. |
| „ 18th | 24 oz. | 1022 | 157·5 grs., or 1·5 p.c. | Neutral; no albumen or blood; mucus much less. | Ditto. |
| „ 19th | 38 oz. | 1025 | 266 grs., or 1·6 p.c. | Alkaline; no albumen; phosphates; mucus. | Ditto. |
| „ 20th | 28 oz. | 1024 | 245 grs., or 2 p.c. | Acid; no albumen or mucus; phosphates. | The same, with chicken jelly. |
| „ 21st (*28th day*) | 21 oz. | 1022 | 220·5 grs., or 2·4 p.c. | Acid; no albumen; some phosphates. | Ditto. |

| | | | | | |
|---|---|---|---|---|---|
| Nov. 22nd | 28 oz. | 1020 | 294 grs., or 2·4 p.c. | Acid; no albumen; lithates and phosphates. | Milk and pudding and chop. |
| „ 23rd | 45 oz. | 1020 | 275·625 grs., or 1·4 p.c. | Strongly alkaline; cloudy with phosphates; quantity of mucus; no albumen. | Chop, milk, egg, chicken broth. |
| „ 24th | 22½ oz. | ... | 177·1875 grs., or 1·8 p.c. | Acid; cloudy; no albumen; phosphates; large quantity of mucus. | The same as yesterday, with rabbit. |
| „ 25th | 24¼ oz. | 1025 | 227·39 grs., or 2·1 p.c. | Acid; no albumen; lithates. | Ditto. |
| „ 26th | 23 oz. | 1022 | 322 grs., or 3·2 p.c. | Acid; clear; no albumen; mucus; lithates; phosphates. | Ditto. |
| „ 27th | 24 oz. | 1030 | 210 grs., or 2 p.c. | Acid; cloudy with lithates; no albumen. | Ditto. |
| „ 28th (*35th day*) | 43 oz. | 1022 | 338·625 grs., or 1·8 p.c. | Acid; no albumen; lithates. | Ditto. |
| „ 29th | 55 oz. | ... | 385 grs., or 1·6 p.c. | Acid; clear; no albumen; no phosphates or lithates. | Ditto. |
| „ 30th | 41 oz. | 1017 | 251·125 grs. or 1·4 p.c. | Acid; clear; no albumen. | Ditto. |
| Dec. 1st | 48 oz. | 1015 | 252 grs., or 1·2 p.c. | Acid; clear; no albumen, phosphates or lithates. | Ditto. |
| „ 2nd | 57 oz. | 1015 | 249·375 grs., or 1 p.c. | Acid; clear; no albumen. | Ditto. |
| „ 3rd | 55 oz. | 1012 | 240·625 grs., or 1 p.c. | Acid; clear; no albumen; light coloured. | Ditto. |
| „ 4th | 40 oz. | ... | 262·5 grs., or 1·5 p.c. | Acid; clear; no albumen. | Ditto. |
| „ 5th (*42nd day*) | 34 oz. | 1025 | 267·75 grs., or 1·8 p.c. | Acid; cloudy; no albumen; few lithates. | Ditto. |
| „ 6th | 67½ oz. | 1012 | 236·25 grs., or ·8 p.c. | Acid; clear; no albumen; light coloured. | Ditto. |
| „ 7th | ... | 1012 | ... | Acid; clear; no albumen; light coloured. | Ditto. |
| „ 8th | 51 oz. | ... | 178·5 grs., or ·8 p.c. | Acid; clear; no albumen. | Ditto. |
| „ 9th | 76 oz. | ... | 166·25 grs., or ·5 p.c. | ... | Ditto. |
| „ 10th | 69 oz. | 1015 | 301·9 grs., or 1 p.c. | Clear; acid; no albumen. | Ditto. |

# ON THE DISTRIBUTION

OF THE

# "TUBERCLE BACILLI" IN THE LESIONS OF PHTHISIS.

BY

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The publication of Koch's memorable paper in the 'Berliner klinische Wochenschrift,' in 1882, has led to a large number of inquiries into the relation of the so-called tubercle bacilli to the process of tuberculosis. It may be safely said that all attempts to discredit the existence of these bacilli, or to assign to them inorganic properties, have entirely failed. Indeed, it would seem rather that there is a disposition to accept this particular micro-organism as a full and sufficient explanation of the whole question of tuberculosis.

Tubercle bacilli, *i. e.* bacilli having the special re-action to certain aniline dyes discovered by Koch, are now known to be present in the pulmonary cavities of all cases of true "tubercular phthisis."

It would also be granted that the same organisms can be detected in the sputum of nearly all cases of this

disease, at some stage or other of the patient's life. They have been found in the lesions of artificial tuberculosis of animals and in acute miliary tuberculosis and phthisis in the human subject. It seems that whereas the bacilli are very numerous in artificial tuberculosis, they are less abundant in what are termed "tuberculous" diseases in man. Under the last heading it is usual now to include not only acute miliary tuberculosis and phthisis, but also scrofulous glandular and joint affections, and even lupus.

The experimental side of the question has been ably investigated in this country by Mr. Watson Cheyne, on the lines laid down by Koch himself.

Mr. Cheyne's report published in the 'Practitioner' of April, 1883, strongly confirms Koch's conclusions. The results of the same observer's examination of a certain number of cases of acute miliary tuberculosis and phthisis agree with Koch's experience. Hitherto, however, the number of observations on the distribution of "tubercle bacilli" in the tissues of man has not been large in England.

It seemed desirable, therefore, in view of the great importance of the subject, to inquire into the distribution of these bacilli in a large number of cases of phthisis.

It may be stated at once that this paper will be confined mainly to the consideration of the anatomical relations of these micro-organisms to the various lesions found in this disease.

No attempt will be made to discuss the etiology of phthisis or tuberculosis, except in so far as any information concerning the local distribution of the "tubercle bacilli" may seem to have a distinct bearing on the question.

The results of the present investigation refer mainly to the lung and in a less degree to the larynx, intestine, and lymphatic glands. Other organs and tissues have been examined in comparatively isolated cases and the results obtained will be given for what they are worth.

In the case of the lung, the object kept in view was to determine the anatomical distribution of the bacilli in the various lesions comprised in the phthisical process; in other words, to find out whether they were present in all the lesions of this disease, and if not, to determine their seat of election.

In describing the anatomical lesions it will be convenient to avoid the use of the term "tubercle" as far as possible, especially in the case of the lung, so as to avoid misconception.

The following list may be said to comprise the main pulmonary lesions of phthisis:

(1) Nodules in size from a millet-seed upwards.
(2) Tracts of consolidation, mostly lobular.
(3) Fibroid induration.
(4) Cavities.

The "nodules" may occur singly or in groups, may be transparent, opaque, or actually softening.

The "tracts of consolidation" may present all stages from commencing catarrhal pneumonia (pinkish consolidation) to complete caseation (caseous pneumonia). Fibrinous matter and small cells are found in some cases in groups of alveoli.

The nodular lesions include bronchioles, alveolar passages and their corresponding alveoli, together with the alveolar walls. Such lesions may be regarded as broncho-pneumonic.

In the case of phthisis and in most instances of acute miliary tuberculosis, this description applies not only to the larger nodules, but also to the true miliary granulation.

The histological characters of these several lesions are too well known to justify a detailed account of them here. It must be borne in mind, however, that in the case of phthisis the very earliest miliary nodules are nearly always associated with a certain amount of fibroid growth. This is found also in the corresponding lesions of acute miliary tuberculosis in some cases, but in a very much

slighter degree. This distinction must not be lost sight of.

The consolidation which has been incidentally described as lobular is sometimes apparently "lobar," and has been so termed by some writers. This, however, is probably seldom the case. A careful examination rarely fails to establish the lobular origin and nature of such consolidation.

The term "cavities" is intended to include true pulmonary and so-called bronchiectatic cavities.

Pigmentation may be associated with any of the foregoing changes.

*Methods employed.*

Hardening was effected by means of alcohol only, except in a few cases, where a mixture of alcohol and chromic acid one sixth per cent. was used.

The sections were stained with the Weigert-Ehrlich solution of fuchsin, then treated with nitric acid, and subsequently stained with methylene blue.

Oil of cloves was used for clearing the sections at first. But since Koch's recommendation of oil of cedar in his complete memoir ("Die Aetiologie der Tuberkulose," 'Mittheil. aus d. Kais. Gesundheitsamte,' Bd. ii), I have invariably used this instead, as the aniline colours are insoluble in it. I have also discarded the ordinary chloroform solution of Canada balsam, and have used instead a solution of balsam in benzol for mounting sections.

In all cases the sections were examined with Abbé's condenser in conjunction with Zeiss's microscope, oculars ii and iv. The lenses used were Zeiss's F, Hartnack's Nos. 4 and 7.

Koch seems to suggest that the use of chromic acid as a hardening agent interferes with the staining of the bacilli. My experience in the six or seven cases that I used chromic acid was that the bacilli were quite as well

stained as when I used absolute alcohol only. I gave up chromic acid, however, for the reason that it interferes with the staining of the tissues with methylene blue. In chromic acid specimens the tissues acquire a faint greenish stain with the blue solution.

The blue staining also is far more perfect when the sections are cleared with oil of cedar instead of oil of cloves.

*Cases.*

1. *Eliza Q.—Chronic phthisis.*—(*a.*) Emphysematous lung studded with nodules of the size of a hemp-seed or rather larger. The nodules consist of aggregations of caseating areas representing groups of alveoli, alveolar passages, and bronchioles. The central parts of the nodules are caseous, the peripheral portions consist of a fibro-cellular zone divisible into an internal and external territory; the latter containing more closely crowded cells of smaller size. Numerous pigmented giant-cells are scattered through the fibro-cellular zone. Connective-tissue development is proceeding in this zone. Some of the nodules contain thickened and dilated bronchioles; in some cases the thickening is mainly of a fibrous nature, in others it consists of a cellular infiltration. Scattered small groups of bacilli are seen in comparatively few nodules, and are situated in caseating alveoli. No bacilli in giant-cells. Some micrococci in caseous matter in a few spots.

(*b.*) Scrapings from the wall of a large cavity contained numerous bacilli.

(*c.*) Scrapings from a partially softened caseous mass contained few bacilli.

(*d.*) Scrapings from a firm caseous part showed no bacilli.

I have described the rough anatomy of the nodules in the present case as being a fairly typical specimen of the ordinary discrete nodular form of the disease. In the remaining cases it may be assumed that the nodules conform in the main to the above type, unless a statement is made to the contrary.

2. *Edwin B.—Phthisis.*—(*a.*) Lung with commencing catarrhal

pneumonia, through which are scattered a few small fibro-caseous nodules. No bacilli in any part.

(*b.*) Emphysematous lung studded with similar nodules. No bacilli found.

3. *John P.—Phthisis.*—(*a.*) Emphysematous lung studded with racemose groups of fibro-caseous miliary nodules. No bacilli found in any part.

(*b.*) Thickened pleura containing fibro-caseous miliary tubercles, with giant-cells. No bacilli found.

4. *Gustav G.—Acute phthisis.*—(*a.*) Fibro-caseous nodules distributed through emphysematous lung. Bacilli in small groups in a few caseous spots, large numbers in the walls of microscopical cavities and in infiltrated brochioles.

(*b.*) Fibro-caseous miliary nodules in thickened pleura. No bacilli found.

5. *James W.—Phthisis.*—(*a.*) Fibro-caseous nodules in slightly emphysematous lung. No bacilli found.

(*b.*) Pleura much thickened, and containing fibro-caseous miliary tubercles. No bacilli found.

6. *Wm. F.—Phthisis.*—Spongy lung studded with small whitish nodules consisting mainly of fibrinous matter blocking up the alveoli, and containing scanty small cells. Some alveoli filled with blood. No bacilli found.

7. *Ellen B.—Phthisis.*—Fibro-caseous racemose nodules in spongy lung. Bacilli scanty, and only found in caseous alveoli here and there.

8. *Jane S.—Phthisis.*—(*a.*) Opaque miliary nodules in œdematous lung, consisting mainly of alveoli filled with fibrinous matter and containing here and there a few large cells with indistinct outline. Bacilli extremely few and scattered in the cellular parts only.

(*b.*) Larynx. Great thickening of aryteno-epiglottic folds, without ulceration. Miliary tubercles in different stages and large-celled infiltration in submucous tissue. Bacilli few and scattered in epithelioid patches.

9. *Henry J.—Chronic phthisis, extensive emphysema, much pigmentation of lung and pleura.*—(*a.*) Pleura. Fibro-caseous miliary tubercles. No bacilli found.

(*b.*) Lung containing similar but larger nodules, in which caseation is less advanced, and traces of cells are visible. Bacilli in some nodules, but as a rule scanty; large numbers in a few spots.

10. *John W.—Phthisis; disseminated miliary tuberculosis.*—(*a.*) Lung containing miliary caseating nodules, with numerous giant-cells in coarsely reticulated zone. No bacilli found in giant-cells,

or in any part of most of the nodules. In a very few cases, two or three bacilli were found in caseating alveoli.

(*b.*) Liver containing numerous fibro-caseous miliary tubercles, with numerous giant-cells. No bacilli found, but, in a few sections some granules of the size of micrococci were found stained red. These granules were not in the tubercles, but were contained in liver-cells in the neighbourhood of tubercles.

(*c.*) Miliary tubercle of pia mater, with scarcely any caseation, in connection with the wall of a small vessel. Bacilli present in considerable numbers among epithelioid cells.

11. *Louisa F.—Phthisis*—Emphysematous lung containing fibro-caseous miliary nodules. Bacilli in small numbers in a few caseous spots, and in larger numbers in microscopical cavities contained in the nodules. A few bacilli in infiltrated bronchioles in some cases.

12. *Elisa H.—Phthisis.*—Lung examined fresh after freezing.—(*a.*) Small fibro-caseous nodules. Bacilli rather few in caseous alveoli, and in surrounding large-celled areas.

(*b.*) Firm, caseous, pneumonic tract. Caseation uniform. No bacilli found.

13. *Amelia B.—Phthisis; pulmonary and laryngeal.*—(*a.*) Tuberculous lymphangitis of lung. A string of miliary nodules attached to either side of the thickened interlobar septum, lung tissue around spongy. Microscopically, the interlobar septum contained caseous tracts with giant-cells, the nodules on either side of it exactly resembling those found in the lung in ordinary cases of phthisis, and consisting of groups of caseating alveoli. Microscopical cavities in nodules here and there. The miliary nodules were evidently secondary to the caseation of the interlobar septum. Bacilli in moderate numbers in some of the nodules where caseation is recent, and in the walls of two microscopical cavities. No bacilli in the old caseous matter of septum, or in the giant-cells of this part.

(*b.*) Larynx. Deep tuberculous ulceration of the inter-arytenoid fold. Scanty fibro-caseous miliary tubercles, and abundant large-celled infiltration. Bacilli in moderate numbers on the ulcerated surface, and in the more superficial tubercles and infiltration. Bacilli seen in one giant-cell.

14. *Alfred F.—Phthisis.*—Emphysematous lung containing small nodules, in which the fibroid tendency is unusually pronounced. No bacilli found.

15. *Henry B.—Phthisis.*—(*a.*) Tracts of catarrhal pneumonia, with scanty caseating foci. No bacilli found.

(*b.*) Small fibro-caseous nodules in spongy lung. No bacilli found.

16. *Eleanor M.—Phthisis.*—Tuberculous lymphangitis. Thickened

pleura, containing miliary tubercles and caseous tracts. Subjacent lung almost entirely spongy, with exception of its extreme subpleural layer, in which there are numerous fibro-caseous miliary nodules continuous with the thickened pleura. These nodules resemble those commonly found in the lung, consisting of infiltrated alveoli and alveolar walls with microscopical cavities in the caseating parts here and there. Bacilli in considerable numbers in the minute cavities. None elsewhere. (See Plate II, fig. 3.)

17. *George F.*—Large caseous mediastinal glands, with subjacent miliary tuberculosis of lung.

(*a.*) Caseous matter from gland, consisting simply of fine detritus, contained no bacilli.

(*b.*) Fibro-caseous miliary nodules in lung. In most cases contained no bacilli; but in a few cases one or two bacilli were seen in alveoli whose cells were beginning to caseate.

18. *Eleanor C.—Phthisis.*—Fibro-caseous miliary nodules in emphysematous lung. Much pigmentation. Bacilli very scanty, in a few caseous spots.

19. *Richard H.—Phthisis; pneumothorax.*—Examined fresh. Lung containing fibro-caseous nodules of various sizes, with much small-celled infiltration of alveolar walls. Bacilli very scanty, and only found in extremely few nodules in caseating alveoli. Micrococci in places in large numbers.

20. *John P.—Phthisis; emphysema.*—(*a.*) Emphysematous lung studded with fibro-caseous miliary nodules. Two different parts examined. (i) Early stage: nodules very scanty. (ii) Later stage: nodules more numerous, and alveolar walls pigmented and infiltrated with small cells. In a very few sections out of a large number bacilli were found in the earlier lesions in caseous spots. None in (ii) later stage.

(*b.*) Firm, caseous suprarenal capsules. Closely crowded caseating areas, with giant-cells and irregular large-celled infiltration. A few bacilli were found in some few caseous spots and in surrounding large-celled zone.

21. *Jeremiah H.—Phthisis; pneumothorax.*—(*a.*) Small fibro-caseous nodules in emphysematous lung. Bacilli in moderate numbers in microscopical cavities in the nodules; less abundant in walls of infiltrated bronchioles. Scattered groups in caseous aveoli, enormous numbers in a few spots close to minute cavities.

(*b.*) Bronchial gland containing caseous areas and miliary tubercles, with giant-cells in their peripheral course of reticulated zone. A few bacilli in scattered caseous spots and in tubercles here and there; rarely more than two bacilli in the same field.

(*c.*) Tubercular ulceration of small intestine. No bacilli found in most sections. One or two bacilli in a few fibro-caseous tubercles in submucosa.

22. *Susan H.—Phthisis.*—Tuberculous lymphangitis of lung, starting from thickened caseous interlobar septum. (Lesions exactly like those in Case 13.) No bacilli found in nodules or in caseous pleura

23. *Thomas C.—Phthisis.*—(*a.*) Spongy lung, with scattered small fibro-caseous nodules. Bacilli scanty, and only in scattered caseous spots in nodules.

(*b.*) Spleen. Amyloid degeneration and irregular caseous patches containing giant-cells. No bacilli found.

(*c.*) Mediastinal glands. Fibro-caseous miliary tubercles, with numerous giant-cells. No bacilli found.

24. *John G.—Phthisis.*—Spongy lung, containing closely set miliary fibro-caseous nodules. Bacilli very numerous in the nodules, among the caseating epithelioid cells, in caseous tracts, in the infiltrated walls of bronchioles, and in small microscopical cavities.

25. *Richmond W.—Acute phthisis.*—(*a.*) Caseous pneumonia. Caseation almost uniform and very firm. Contents of alveoli fibrinous in many parts. Scarcely a trace of cellular structure in most parts. No bacilli could be found after careful examination of different parts.

(*b.*) Tubercular ulceration of small intestine. Miliary tubercles and large-celled infiltration in submucosa; tubercles mostly recent. Bacilli very numerous, and widely distributed in tubercles and epithelioid cells; more abundant in tubercles. Bacilli in a few giant-cells.

26. *Case of phthisis*, sent by Dr. Powell.—Extensive catarrhal pneumonia, with irregular caseating patches. Bacilli few, and only in scattered points where caseation is incomplete. No bacilli where caseation is advanced.

27. *Emma W.—Phthisis.*—Early caseous pneumonia of very irregular character. In some parts catarrhal pneumonia; elsewhere caseous patches, in which are scattered softening foci. Bacilli very irregularly distributed. The walls of microscopical cavities contain enormous numbers. In most sections two or three large groups are seen in caseous alveoli and interalveolar spaces, none in catarrhal pneumonic parts. The bacilli are not widely disseminated, but seem to be exclusively collected in large groups. Walls of small veins in places infiltrated with epithelioid cells, among which bacilli were seen in a few instances.

28. *Stephen H.—Phthisis; pneumothorax.*—(*a.*) Caseous pneumonia like that in last case. Much catarrhal pneumonia. Bacilli

in small numbers, but widely diffused through parts where caseation is early.

(*b.*) Mediastinal gland containing miliary tubercles and epithelioid areas. Caseation commencing at various points. Bacilli numerous both in tubercles and in large-celled infiltration, especially in cortical portion of gland where the disease is most marked.

29. *Emily P.—Phthisis.*—(*a.*) Caseous pneumonia; caseation irregular; much catarrhal pneumonia. Bacilli scattered in small numbers where caseation is less advanced, collected in groups in a few instances in caseous parts.

(*b.*) Caseous pus from Fallopian tubes, consisting simply of fine detritus without any cell structure. No bacilli.

30. *Edith G.—Phthisis.*—Irregular caseous pneumonia resembling last case. Bacilli in enormous numbers, collected in groups in caseous parts. None in catarrhal pneumonic patches.

31. *Emily G.—Phthisis.*—(*a.*) Caseous pneumonia. Caseation firm and uniform. Contents of alveoli fibrinous in some parts. Cellular elements in most sections absent or very scanty. No bacilli could be found although numerous sections were taken from different parts.

(*b.*) Larynx. Epiglottis enormously thickened and only very slightly ulcerated. Numerous miliary tubercles in various stages in submucosa, with abundant giant-cells; also extensive large-celled infiltration. Bacilli very few in tubercles and in the base of the ulcers.

32. *Kate G.—Phthisis.*—Caseous pneumonia. Caseation very firm, but less uniform than in last case, with scattered patches of catarrhal pneumonia. No bacilli found in caseous parts. One group, however, found at margin of caseation in an alveolus containing an increased number of epithelial cells.

33. *Fred. H.—Lympho-sarcoma of Mediastinal Glands invading the lung, which contained also crops of miliary nodules, irregularly pigmented patches of greyish consolidation resembling caseous pneumonia, and some small cavities.*—(*a.*) Mediastinal glands showed the usual lympho-sarcomatous structure, and contained no bacilli.

(*b.*) Pericardium thickened, and containing caseating tracts. Bacilli in a few large groups among caseous material.

(*c.*) Lung. 1. The contents of small cavities contained extremely few bacilli.

2. Large lymphomatous nodules showed same structure as mediastinal glands, and were free from anything like caseation. No bacilli.

3. Greyish consolidated parts showed the structure of caseous

pneumonia combined with much catarrhal pneumonia. Bacilli in great numbers in various parts when caseation is early among epithelioid cells, and in microscopical cavities. A few also in infiltrated walls of bronchioles.

4. Miliary nodules consist of groups of caseating alveoli and bronchioles exactly like miliary tubercles. No bacilli found in these nodules.

34. *George B.—Acute miliary tuberculosis.*—(*a.*) Lung with varying amount of catarrhal pneumonia stuffed with enormous numbers of slightly opaque miliary and submiliary nodules, and scattered fibro-caseous nodules of larger size. In nearly all cases the nodules obviously consisted of groups of alveoli filled with a finely granular material, in which were traces of cells in some instances. Here and there an alveolus was filled with large epithelial cells. Frequently an infiltrated caseating bronchiole was seen in the midst of the nodule or at one side of it. A large-celled infiltration of the walls of small veins was seen in some sections. Groups of bacilli were found in a few of the larger caseous nodules, but none were detected in the smaller ones except in the caseating walls of some of the bronchioles. Numerous sections from different parts of the lung gave the same result.

(*b.*) Spleen. Numerous fibro-caseous miliary tubercles with giant-cells. No bacilli found.

(*c.*) Kidney. Cortex contained scanty caseating miliary nodules and irregular patches of small-celled growth in which were traces of tubules and glomeruli. Bacilli few, and only in caseous spots.

35. *Jessie H.*, æt. 5½.—*Acute miliary tuberculosis* (specimens sent by Dr. Angel Money).—(*a.*) Lung with irregular catarrhal pneumonic tracts, studded with rather scanty caseating nodules of the size of hemp-seed. The nodules consist of groups of alveoli filled with caseating epithelioid cells, infiltrated bronchioles, and an irregular peripheral small-celled zone; giant-cells scanty. Centres of nodules completely caseous in places. Bacilli present in enormous numbers in almost every single nodule, especially among epithelioid cells, also in giant-cells, bronchioles, and in smaller numbers in older caseous spots. No bacilli in small-celled zone, or in surrounding catarrhal pneumonic parts. In one place the walls of a minute artery were seen to be infiltrated with epithelioid cells, which gave rise to a nodular projection from the intima into the lumen of the vessel, which was patent and filled with blood-corpuscles. Bacilli were present in great numbers among the epithelioid cells in the vascular wall. (See Plate I, figs. 1 and 2.)

(*b.*) Liver. Scanty miliary tubercles. Bacilli in rather small numbers in giant-cells, and among the epithelioid cells.

(*c.*) Spleen. Numerous miliary tubercles. Bacilli in large numbers among epithelioid cells and in giant-cells.

36. *Hubert F. L.*, æt. 3.—*Acute miliary tuberculosis* (specimens sent by Dr. Angel Money).—(*a.*) Lung. Irregular patches of catarrhal pneumonia and small caseous nodules and tracts. Bacilli in large numbers in caseating tracts, comparatively few in nodules, none in catarrhal pneumonic parts.

(*b.*) Kidney. Scanty miliary tubercles, in which there are traces of caseation, and islets of small-celled infiltration in cortex. No bacilli found.

(*c.*) Liver. Miliary tubercles. Bacilli very few in giant-cells, among epithelioid cells, and in caseating foci.

37. *Case of acute miliary tuberculosis in a child* (specimens sent by Dr. Chaffey).—(*a.*) Lung containing scattered nodules as large as hemp-seed. Nodules mainly consist of alveoli filled with fibrinous-looking material, very few large cells, and scanty giant-cells. Bacilli in great numbers in alveoli, few in giant-cells.

(*b.*) Liver. Extreme fatty degeneration and scanty miliary tubercles, some recent, most of them fibro-caseous. Bacilli few in giant-cells, among epithelioid cells, and, in a few cases, in more recent caseous foci.

38. *Case of acute miliary tuberculosis in a child* (another case sent by Dr. Chaffey).—Lung thickly studded with miliary nodules, most of which are undergoing a fibro-caseous change. Bacilli few and scattered, among epithelioid cells, in some giant-cells, and in caseating spots.

39. *George H.—Acute miliary tuberculosis.*—(*a.*) Lung œdematous and studded with miliary caseating nodules. Bacilli in small numbers and scattered in early caseous spots, where traces of epithelioid cells are to be seen. None in surrounding tracts of catarrhal pneumonia.

(*b.*) Kidney. Scattered submiliary tubercles. Bacilli in rather small numbers in most of the tubercles, among epithelioid cells, and in caseating foci, groups in some cases, Some Malpighian capsules contain a finely granular crescentic zone partially surrounding the degenerate glomerulus, the whole capsule being encircled by an outer epithelioid zone. Bacilli in varying numbers in the crescentic internal amorphous (caseating) zone, and in the peripheral epithelioid ring.

(*c.*) Liver. Numerous submiliary tubercles. Bacilli few and scattered among epithelioid cells, none in giant-cells, which are scanty.

(*d.*) Spleen. Submiliary tubercles. Bacilli very few and scattered among epithelioid cells and in a few giant-cells.

40. *Amos P.—Acute miliary tuberculosis.*—Lung containing evenly distributed miliary tubercles, in which caseation is beginning in nearly all cases. Bacilli few in giant-cells and among epithelioid cells.

41. *Geo. C.—Phthisis.*—Thick fibroid wall and trabecula of large cavity. Bacilli in moderate numbers in superficial caseous layer in both positions. None in deeper parts.

42. *Eli M.—Phthisis.*—(*a.*) Fibroid wall of small suppurating cavity. No bacilli found in sections, in consequence probably of superficial layer having been detached in preparation, for a scraping from hardened cavity wall showed abundant bacilli.

(*b.*) Tubercular ulceration of small intestine in early stage. No bacilli found in most sections. One or two bacilli in a few fibro-caseous tubercles.

(*c.*) Mediastinal gland containing firm caseating patches with giant-cells. No bacilli found.

43. *James M.—Phthisis.*—Thick fibroid wall of small cavity. No bacilli seen in cavity wall in sections. A scraping from the cavity wall after hardening showed a few bacilli.

44. *Hugh C.—Phthisis.*—(*a*). Pleura thickened, and containing fibro-caseous miliary tubercles with numerous giant-cells and caseating tracts. No bacilli found.

(*b.*) Trachea. Superficial ulceration with necrotic caseating surface. Small-celled infiltration of mucosa and round about mucous glands and ducts. No miliary tubercles or large-celled growth. No bacilli in the tissues; one or two are seen on the ulcerated surface.

45. *Edward S.—Phthisis.*—Pleura thickened and containing caseous tracts. No bacilli found.

46. *James L.—Sacculated bronchiectasis; abscess of brain.*—Extensive excavation of one lung, with foul, inspissated, secretion-like softening caseous matter. Bronchi generally dilated. No "tuberculous" lesion.

(*a.*) Fœtid purulent expectoration during life contained no bacilli, but numerous micrococci.

(*b.*) Discharge from cavity after tapping during life contained micrococci, but no bacilli.

(*c.*) Contents of cavities after death contained micrococci, but no bacilli.

(*d.*) Pus from cerebral abscess contained micrococci, but no bacilli.

47. *Charles H.—Sacculated bronchiectasis; abscess of brain.*—Cavities in right upper lobe with contents like those in last case. Bronchi generally dilated. No "tuberculous" lesion.

(*a.*) Cavity contained micrococci, but no bacilli.

(*b.*) Pus from cerebral abscess gave a similar result.

48. *Charles L.—Aneurysm of aorta, compressing left bronchus.*—Excavation of left lung (bronchiectatic). Cavity contents contained some micrococci and a few putrefactive bacilli, but no "tubercle bacilli."

49. *Margaret S.—Syphilitic stricture of bronchus.*—Excavation of corresponding lung (bronchiectatic). Cavity secretions contained no "tubercle bacilli." A few micrococci.

50. *Archibald M.—Malignant disease of lung; sarcoma.*—Nodules of all sizes and small cavities containing blood-stained grumous fluid.

(*a.*) Cavity secretions contained no "tubercle bacilli."

(*b.*) Nodular growths in lung contained no bacilli.

51. *Walter A.—Laryngeal phthisis.*—Tubercular ulceration and great thickening of aryepiglottic fold. Tubercles scanty. Extensive large-celled infiltration. Bacilli scattered in small numbers on the floor of the ulceration and in the submucous tubercles and epithelioid patches.

52. *Charles L.—Laryngeal phthisis.*—Tuberculous ulceration of larynx and trachea. Small tumours apparently situated on vocal cords, but really springing from lateral aspect of interarytenoid fold. Bacilli in large numbers in the tubercles and epithelioid areas, in arynx and trachea, and even more abundant in the small tumours, which consist of collections of miliary tubercles with intervening epithelioid areas.

53. *John R.—Phthisis.*—Larynx. Tubercular ulceration of interarytenoid fold. Numerous caseating miliary tubercles, and large-celled growth in mucosa and submucosa. Bacilli present in very scattered spots and in small numbers at the surface, and in the more superficial tubercles and epithelioid areas.

54. *Wm. J. T.—Phthisis.*—(*a.*) Larynx. Deep tubercular ulceration of the interarytenoid fold, exposing the muscular layer. Bacilli in enormous numbers on the floor of the ulceration and in the subjacent tubercles and epithelioid areas.

(*b.*) Intestine. Deep tubercular ulceration of ileum. Bacilli numerous at ulcerated surface and in submucous tubercles. Some bacilli in places in subserous tubercles.

55. *Wm. F.—Phthisis, pulmonary and laryngeal.*—Larynx. Miliary nodules, without ulceration in their immediate vicinity, in the

"sinus pyriformis," consisting mainly of small-celled growth, with scattered, faintly staining, large-celled areas. A very few bacilli were found in the latter position in a few sections only.

56. *George G.—Phthisis, pulmonary and laryngeal.*—Larynx. Tubercular ulceration, with great fibroid thickening, both in the base of the ulcers and in their neighbourhood. Bacilli extremely scanty in tubercles here and there.

57. *Thomas C.—Phthisis, pulmonary and laryngeal.*—Larynx. Great swelling of epiglottis, with scarcely any ulceration. Miliary tubercles and large-celled infiltration. Bacilli widely diffused in large numbers in tubercles and large-celled patches. Many giant-cells contain bacilli.

58. *John P. J.—Laryngeal phthisis.*—(*a.*) Tuberculous ulceration of larynx and trachea. Tubercles recent in larynx; fibro-caseous in trachea; epithelioid areas. Bacilli scattered in very small numbers through tubercles, and large-celled infiltration in larynx and trachea.

(*b.*) Tuberculous ulceration of cæcum. Bacilli few and scattered in caseating tubercles and in tubercles of reticulated type.

(*c.*) Mesenteric gland, containing recent miliary tubercles and larger fibro-caseous nodules. Bacilli scattered in small numbers through both varieties of nodule.

59. *Frederick J.—Laryngeal phthisis.*—(*a.*) Pharynx. Tubercular ulceration. 1. Small superficial ulcers with subjacent miliary tubercles and epithelioid areas. Bacilli scattered in small numbers in tubercles and large-celled patches.

2. Large sharply-cut ulcers with much fibroid growth in their bases. Scanty miliary tubercles of fibro-caseous structure. No bacilli found.

(*b.*) Tubercular ulceration of larynx. A small tumour growing from interarytenoid fold amidst the ulceration, and consisting of miliary tubercles and epithelioid areas. Bacilli numerous in both lesions of tumour.

60. *Elisabeth S.—Phthisis.*—Tuberculous ulceration of small intestine. Bacilli very few, and found in a comparatively small number of sections in the floor of the ulceration and in fibro-caseous submucous tubercles. In a few instances groups of small spherical granules like micrococci stained red were found in the submucosa. These bodies appeared to be situated in fat-cells. In the same sections other fat-cells contained groups of somewhat similar granules stained blue.

61. *William H.—Phthisis.*—Tubercular ulceration of small intestine with numerous fibro-caseous miliary tubercles in the subserous tissue. Great atrophy of intestine. No bacilli found except in one

section, where a caseating submucous tubercle contained one well-marked bacillus.

62. *Janet W.—Phthisis.*—Intestine. Superficial ulceration and cellular infiltration of mucous coat. No tubercles or "tuberculous" structure. Lymphatics stuffed with small cells. Fibroid induration where the disease is most advanced. Great atrophy of intestine. No bacilli found.

63. *Henry B.—Phthisis.*—Intestine. Tubercular ulceration in different stages in different parts. No bacilli found in early or advanced ulceration.

64. *Emily C.—Phthisis.*—Intestine. Deep tubercular ulceration exposing muscular layer. Submucous tubercles. Bacilli fairly numerous at the ulcerated surface and in caseating tubercles.

65. *John M.—Phthisis.*—Superficial ulceration of small intestine, with abundant small-celled infiltration of mucosa and submucosa. Fibroid thickening of base of ulcers in places. No tubercles or epithelioid areas. No bacilli.

66. *James B.—Phthisis.*—(*a.*) Superficial ulceration of small intestine, with small-celled infiltration of mucosa and submucosa. No tubercles or epithelioid areas. No bacilli found.

(*b.*) Similar infiltration, but no ulceration. No bacilli found.

Great atrophy of intestine in both cases.

67. *John H.—Abscess of liver.*—Ulceration of large intestine with enormous fibroid thickening of gut, probably due to old dysentery. Small-celled infiltration of the whole intestinal wall, with widely disseminated micrococci, but no bacilli.

68. *John H.—Phthisis.*—Bronchial gland containing caseating areas and fibro-caseous miliary tubercles, with giant-cells. No bacilli found.

69. *Annie E.—Phthisis.*—Bronchial gland. Similar disease to that in last case. No bacilli found.

70. *Daniel E.—Phthisis.*—(*a.*) Mesenteric gland. Caseation commencing in most parts, numerous miliary tubercles and epithelioid tracts. Bacilli in enormous numbers in all lesions in every single section examined (see Plate II, fig. 4).

(*b.*) Liver. Fatty degeneration, early cirrhosis and miliary tuberculosis. Bacilli in fair numbers in giant-cells and among epithelioid cells of tubercles.

71. *Samuel L.—Phthisis.*—Mesenteric gland in state of uniform firm caseation. No bacilli found.

72. *Henry P.—Phthisis.*—Mediastinal gland containing caseating areas and fibro-caseous miliary tubercles. Bacilli extremely scanty in tubercles.

73. *Arthur T.—Phthisis.*—Tracheal gland. Caseating areas and miliary tubercles, some recent, others fibro-caseous. Bacilli in very small numbers in few sections in tubercles and caseous patches.

74. *George B.—Aneurysm of Aorta.*—Recent miliary tuberculosis of pleura, mediastinal glands, and peritoneum.

(*a.*) Mediastinal gland. Miliary tubercles containing giant-cells and epithelioid areas. No bacilli found.

(*b.*) Peritoneum. A transparent miliary tubercle crushed out and examined in fresh state. Bacilli few around epithelioid cells.

75. *Henry P.—Phthisis.*—(*a.*) Tongue. Tubercular ulceration. Submucous and intermuscular miliary tubercles and epithelioid areas. Bacilli in considerable numbers in the floor of the ulceration and in the more superficial tubercles and large-celled infiltration. In a few spots the bacilli were very numerous.

(*b.*) Caseous abscess of sacro-iliac joint. Bacilli rather few in caseous matter.

76. *Thos. A.—Phthisis.*—Tubercular ulceration of tongue. Very abundant miliary tubercles and large-celled infiltration in submucous and intermuscular tissue. Caseation beginning in some of above lesions. Fibroid induration in submucosa. Bacilli in enormous numbers in both forms of lesion, both in superficial and deeper parts.

77. *John P.—Phthisis.*—Tongue. Small tubercular ulcer. Miliary tubercles with giant-cells, epithelioid areas and caseating patches in subepithelial layer, and between muscular bundles. Bacilli very numerous in all the lesions.

78. *A case of phthisis.*—Transparent miliary tubercle of peritoneum examined fresh. Bacilli few, among epithelioid cells.

79. *A case of phthisis.*—Miliary tubercle of peritoneum. Examined in fresh state. Contained a few bacilli.

80. *Andrew M.—Phthisis.*—Thickened pericardium containing scanty miliary tubercles. Some tubercles fibro-caseous, others of reticulated type. One well-marked bacillus found in a tubercle of latter class. No bacilli elsewhere in numerous sections.

81. *John C.—Phthisis.*—Kidney containing small, irregular, caseating patches in cortex, and small-celled foci. Bacilli few and only in caseous spots.

82. *Charles D.—Phthisis.*—Caseous masses in kidneys. Smaller masses shreddy, larger ones putty-like. Bacilli very numerous in shreddy parts; very scanty in older putty-like material.

83. *Phthisis.*—Tubercular meningitis. Miliary tubercles examined fresh. Bacilli in fair numbers amid the epithelioid cells of perivascular tubercles.

84. *Phthisis.*—Tubercular meningitis. Perivascular miliary tubercles, in which caseation was beginning, contained considerable numbers of bacilli among epithelioid cells.

85. *Geo. A.—Phthisis.*—Tubercular meningitis. Sections of hardened "pia mater" showed well-marked perivascular miliary tubercles, with commencing caseation in most cases. Bacilli in fair numbers both in recent and old tubercles.

86. *Charles H.—Abscess of liver.*—Vesicula seminalis on one side caseous. Bacilli in moderate numbers in soft caseous contents.

87. *John F.—Phthisis.*—Bladder. A group of small circular superficial ulcers. Cellular infiltration of mucous coat. No tubercles. Some few bacilli stained blue in base of ulcers. In one section only a minute caseous nodule was seen embedded in the edge of an ulcer. This nodule contained two or three well-marked red bacilli.

88. *Jane M.—Phthisis.*—Fallopian tube distended with firm caseous matter. Bacilli extremely scanty in caseous contents.

89. *Mary Josephine B.—Phthisis.*—Uterus with caseous degeneration of its mucous membrane and irregular ulceration. A few bacilli found in caseous lining.

90. *Ellen S.—Phthisis.*—Scrofulous disease of tarsus and of kidney. (*a.*) Puriform matter from diseased tarsus contained very numerous bacilli. N.B.—The joint had not been opened, and there was no communication with the atmosphere.

(*b.*) Caseous masses in kidney contained numerous bacilli.

The results of the examination of the tuberculous cases may be thus stated:

| | | | | | | |
|---|---|---|---|---|---|---|
| Lung | . | 31 | cases of | nodular lesion. | Bacilli found in | 24 |
| „ | . | 10 | „ | of caseous pneumonia. | „ | 7 |
| Larynx } Trachea } | . | 13 | „ | ... ... | „ | 12 |
| Intestine | . | 12 | „ | ... ... | „ | 8 |
| Lymph. glands | | 13 | „ | ... ... | „ | 6 |
| Pia mater | . | 4 | „ | ... ... | „ | 4 |
| Peritoneum | . | 3 | „ | ... ... | „ | 3 |
| Pericardium | . | 2 | „ | ... ... | „ | 2 |
| Tongue | . | 3 | „ | ... ... | „ | 3 |
| Liver | . | 6 | „ | ... ... | „ | 5 |
| Kidney | . | 6 | „ | ... ... | „ | 5 |
| Spleen | . | 4 | „ | ... ... | „ | 2 |
| Scrofulous joint | | 2 | „ | ... ... | „ | 2 |

Bacilli were also found in single cases in the vesicula

seminalis, Fallopian tube, uterine cavity, bladder, and suprarenal body.

With reference to the intestine it must be remembered that in three out of the four cases in which no bacilli were found, there were no "tuberculous" lesions in the base of the ulcers.

In the account of the various cases no detailed record has been given of the examination of the contents of pulmonary cavities. I have never yet failed to find bacilli in the cavity secretions of "tuberculous" cases in a large number of observations. They were most plentiful in vomicæ containing thick caseous secretion, and where the cavity appeared to be of recent formation or was undergoing rapid extension.

Where the secretions were of a thin liquid nature bacilli were less numerous. It would seem that these organisms are practically confined to the superficial, caseous, or suppurating lining of the large cavities. This layer is never without bacilli, and indeed mostly teems with them. If, however, this layer be detached, as may easily occur in making sections, no bacilli may be found; in other words, the bacilli do not penetrate far into the cavity wall. This is what might have been expected, as the fibroid growth in these parts must oppose a barrier to the spread of the bacilli.

In microscopical cavities, on the other hand, where we are able to observe the earliest stages in the process of excavation, bacilli are often found both at the margin of the cavity and extending in large tracts into the surrounding caseous tissues. In such cases no demarcating fibroid zone or capsule has developed round these minute cavities.

Four cases of extensive excavation, probably of bronchiectatic origin, in which one lung was alone affected, and no tuberculous lesions were present, supplied the strongest proof of the diagnostic value of the bacilli in determining whether lung disease is tuberculous or not. In one of these cases repeated examination of the expec-

toration and of the cavity contents evacuated by puncture of the chest during the patient's life strongly pointed to a disease of a non-tuberculous character.

This conclusion was fully justified at the autopsy by the general appearance of the lung and by the results of a microscopical examination of the contents of the pulmonary cavities. In each case tubercle bacilli were absent, though abundant micrococci were found.

In the other cases similar disease of the lung was found post mortem, *i. e.* cavities with no tuberculous lesion. The cavity contents contained micrococci, but no tubercle bacilli. Here one would think were conditions as favorable as any likely to arise in the human body for the cultivation of micro-organisms. Both patients during their life were placed in wards occupied by phthisical subjects, so that every opportunity was afforded to the tubercle bacilli to take root in the stagnant cavity secretions of these two men. Apparently the soil was not suited for the nurture and development of these organisms, and this observation suggests that very special conditions are required for the entrance and growth of the tubercle bacillus.

Returning now to the strictly tuberculous cases, we find that the bacilli are present in most cases of nodular lesion, but in the case of phthisis their number is as a rule extremely small.

The explanation of this fact may be in part derived from the almost constant association of fibroid growth even with the earliest nodules. It seems possible that although the presence of bacilli is connected with the development of each nodule in the first instance, yet the subsequent fibrosis and caseation may lead to arrest of development or perhaps to destruction of the micro-organisms. What determines the predominance of fibrosis in one case and softening in another it is hard to say. It is true, no doubt, as a rule that in children and young persons softening predominates over fibrosis, the latter process attaining its highest development in persons of more advanced age.

Or to put it in another way, tissues that are undergoing active growth afford a more luxuriant soil for the development of the bacilli than tissues which are mature or degenerate. Exceptions, however, are not wanting to such general statements, and it is only too plain that the idiosyncracies of tissues in different people are a complete mystery to us.

Whether the final softening of the nodule is the result of an awakened activity of the dormant bacilli or their spores, or whether it is due to a subsequent invasion of these organisms from the air-passages, is a difficult question. It is important to remember the fact, so strongly insisted upon by Koch, that the spores are extremely resistant bodies. Koch found that in certain cases caseous matter apparently containing no bacilli was still infective, and when inoculated set up tuberculosis with a copious development of bacilli. He supposes that in such cases the caseous matter contained the spores, *i. e.* potential bacilli, which only required a suitable nidus for their evolution into mature bacilli. These facts may supply the explanation of the absence of the bacilli in so many of the nodules. It is, however, difficult to account for the rapid disappearance of these organisms from the nodules. I have over and over again examined the earliest miliary granulations I could find in cases of phthisis and always with the same result. Bacilli were always few, and often absent. In the larger nodules, however, where caseation was advanced, bacilli were collected in large groups in many instances.

On the other hand, in the seven cases of acute miliary tuberculosis examined, tubercle bacilli were found in every instance. This is a striking contrast, but I believe it may be explained by the acute nature of the process in this affection as compared with the chronicity of phthisis. It is an interesting fact that out of these seven cases of acute miliary tuberculosis bacilli were only found in extremely large numbers in four, all of which were children. The other three cases were adults.

With reference to the localisation of the bacilli in the structures comprising the nodule, I have found them in most cases only in what seemed to be caseous alveoli or in alveoli filled with large cells, the so-called epithelioid cells.

In the latter case, these large cells stain only slightly and their outlines are very indistinct, an evidence of the advent of caseation. The bacilli were found in the peripheral fibro-cellular layer in very few instances, and only then in comparatively small numbers. In several such instances they were contained in giant-cells.

In a few cases I met with a distinct tuberculosis associated with the presence of bacilli in the walls of small pulmonary veins, and in one instance an arteriole was similarly affected. Such facts strongly support the views of Weigert as to the path by which the tubercular virus commonly enters the circulation.

I have not succeeded in finding the bacilli in the perivascular and peribronchial small-celled growths. They were present often in large numbers in minute microscopical cavities in the nodules, and to a less extent in the infiltrated lining membrane of the corresponding bronchioles.

There can be little doubt that the bacilli are disseminated in part by the lymphatics of the lung. But the prevailing nodular character of the more recent pulmonary lesions suggests that infection of distant parts is the result of inhalation.

In the first case, infection probably takes place from atmospheric sources, as Koch believes, whereas in the later stages this must be largely supplemented by aspiration into the lung of the contents of the cavities.

It is equally difficult to explain the capricious localisation of bacilli in the caseous pneumonic tracts. I can only suggest a similar explanation to that offered in the case of the nodular form. In some of the most severe cases of phthisis, caseous pneumonia is the predominant lesion. It seems doubtful whether any cases of phthisis

commence as a massive pneumonic consolidation. At any rate the pre-existence of a nodular stage can hardly be excluded. If we consider that the primary local manifestation of the disease is nodular it requires no great stretch of imagination to regard widespread consolidation, like caseous pneumonia, as secondary to the presence of discrete nodules. There is ample evidence that this is frequently the sequence of events in the later stages of the disease. Or it may be, as Mr. Watson Cheyne suggests, that in caseous pneumonia the virus is inhaled into the individual alveoli. If this were the case we should expect to find bacilli distributed widely through the consolidation. This, however, has not been my experience. It may be that caseous pneumonia is set up in another way. In certain cases where cavities or rapid softening are present we are reminded that septic processes are going on, not only by the character of the attendant pyrexia, but often also by the odour of the patient's breath. The products of this process are probably gaseous to some extent, and can hardly fail to be inhaled into the alveoli. This may play some part in the production of those large caseous patches so often found in the sternal region in cases of advanced destruction of lung.

I have been unable to find any bacilli in those parts of the lung that have undergone a dense fibroid change. Neither have I succeeded in detecting their presence in the pigmented giant-cells often enclosed in the fibroid tissue, except in the outer fibro-cellular zone of the nodular growths. Here I have found them in several cases. It may now be asked whether there is any evidence that the evolution of each individual nodular lesion of phthisis is invariably associated with the presence of the tubercle bacilli. The answer, I believe, must at present be, that although this is highly probable direct proof is wanting.

It is probable that the presence or absence of these organisms is closely connected with the stage of development of the nodules. The presence of comparatively

In the thirteen cases of intestinal ulceration examined, bacilli were found in eight cases only.

In four cases bacilli were plentiful.

Three of the cases which gave a negative result as regards bacilli presented no typical tuberculous changes, and although the ulceration was slight the mucous coat was greatly atrophied.

In the fourth case, the ulceration was probably due to old dysentery and was in no sense tuberculous. No bacilli were found.

In all the other cases the disease was distinctly tuberculous.

Of the thirteen cases in which lymphatic glands were examined the bacilli were plentiful in three only; in three other cases they were very scanty. In seven cases none could be found.

It seems remarkable at first sight that the mediastinal glands contained so few bacilli. But it is worthy of note that all the glands examined were those of adults, most of whom were dwellers in towns. It is possible that the pigmentation and induration which is so common in the mediastinal glands of such persons renders them less suited for the growth of the bacilli.

The abundant pigment in the glands may possibly obscure the presence of the bacilli to some extent when they are very scanty. The number of bacilli found in these and other glands depends mainly, however, I believe, on the stage of the disease. When the tuberculosis is quite recent they are abundant; in the later stages it may be impossible to discover any at all. The observations referring to other organs than the lung, air-passages, intestine and lymphatic glands are too scattered and few to be of much value.

But the detection of the bacilli in such widely different places as the peritoneum, pia mater, kidney, liver, spleen, tongue, caseous joint and vesicula seminalis, Fallopian tube, uterus, and suprarenal capsule is worthy of note.

It is impossible to doubt that the presence of certain

bacilli is indissolubly bound up with the process of "tuberculosis," although their distribution appears at present to be somewhat capricious.

In pulmonary cavities and in softening caseous matter, wherever it may occur, so long as it be associated with "tuberculous" or scrofulous affections, there we may be sure of finding Koch's bacilli.

It is probable also that in all lesions termed "tuberculous," the same bacilli can be found at certain stages of their development by careful search, if a sufficient number of sections be examined.

But although the extremely small and even insignificant number of these organisms found in many cases of advanced and wide-spread "tuberculous" disease may be to some extent explained, it will appear to many, perhaps, improbable that the presence of a few bacilli can be the sole cause of such extensive structural changes.

I would, however, express the belief that as we know more of the various conditions incidental to the life-history of organisms like the tubercle bacillus, we shall be able to account for much that at present looks like irregularity and caprice in the behaviour of these parasites.

On the other hand it is much to be hoped that Koch's brilliant and valuable discovery may not entirely divert attention from the important influence of constitutional disposition or diathesis in the production of the various affections now grouped together as tuberculous.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 303.)

## DESCRIPTION OF PLATES I AND II.

"Tubercle Bacilli" in the Lesions of Phthisis. (PERCY KIDD, M.A., M.D. Oxon.)

FIG. 1.—*Acute miliary tuberculosis.* Case 35.—Lung: two alveoli which have become fused together. Alveoli filled with large epithelioid cells. Tubercle bacilli in large numbers between the cells, and in a few instances within the cells (*a*, *a*). × 400.

FIG. 2.—*Acute miliary tuberculosis.* Same case.—Lung: arteriole from one of the nodules. Walls of vessel thickened and infiltrated, with epithelioid cells scattered through a finely granular substance (commencing caseation). Calibre of vessel encroached upon by the growth. Vessel still patent, and occupied by blood-corpuscles.

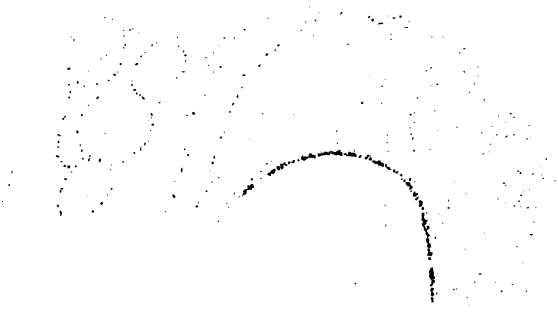
*a*. Thickened wall of vessel.
*b*. Remains of muscular coat.
*c*. Cavity of vessel filled with red corpuscles, and containing a few leucocytes stained blue. × 200.

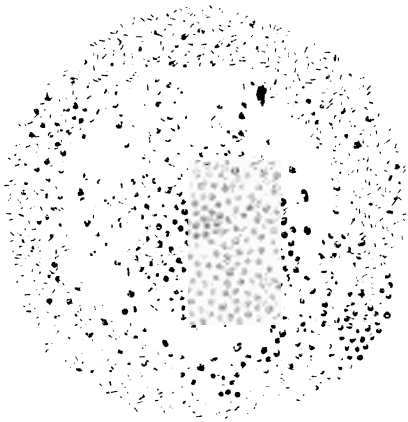
FIG. 3.—*Phthisis.* Case 16.—Lung: section from wall of minute cavity. Inner margin of cavity teeming with bacilli. No bacilli elsewhere.

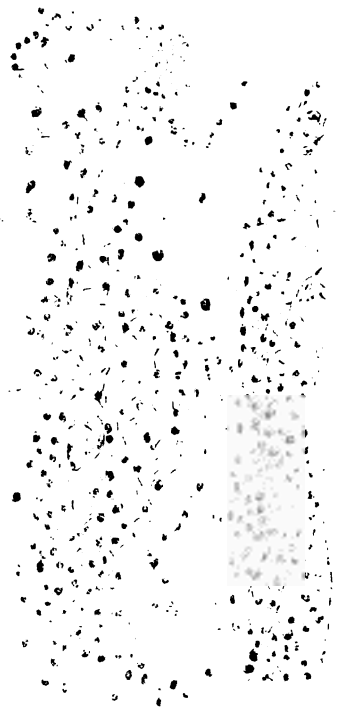
*a*. Cavity.
*b*. Bacillary margin.
*c*. Surrounding caseous tissue. × 75.

FIG. 4.—*Phthisis, with intestinal ulceration.* Case 70.—Mesenteric gland. Follicle from gland containing numerous bacilli, and surrounded by a broad caseous zone teeming with bacilli. The follicle has separated from the caseous zone in the process of preparation.

*a*. Follicle.
*b*. Caseous zone.
*c*. Space due to shrinking of follicle from surrounding zone. × 200.







# CASES

IN WHICH

# PERFORATION OF THE MASTOID CELLS

# IS NECESSARY.

BY

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(Received October 23rd, 1884—Read January 27th, 1885.)

IN the following remarks no reference will be made to the very common instances in which, after inflammation of the middle ear, the usual signs of abscess in the mastoid process, with softening of the external plate of bone, are present; these, especially in the case of children, are so often met with and treated by a free opening on the ordinary principles of surgery, that they are perfectly familiar to everyone. There are, however, a certain number of cases from which the usual appearances, symptoms, and local conditions are absent. Among such may be classed especially those in which the external plate of bone is healthy, but pus has formed within the cells (with its attendant dangers to life), and others in which, although the external plate of bone is the seat of caries, purulent matter is not within the cells but collects elsewhere.

In a previous contribution to this Society on 'Disease of the Mastoid Bone' (vol. lxii of the 'Transactions,'), the conditions under which a perforation should be made into the mastoid cells were discussed, especially in regard to those cases where some of the usual evidences of pus at no great distance from the surface were absent, for where these are present the necessity of this proceeding is universally admitted. The indications for operation in cases of purulent discharge from perforation of the tympanic membrane included the following symptoms: continuous pain in the mastoid region, with œdema on deep and prolonged pressure, pain increased by the recumbent position, a high temperature, and a severe rigor.

Further experience since 1878 has not only confirmed the views which I then expressed, but has forced upon me the conviction that the conditions under which relief may be afforded, and a fatal termination avoided, are more numerous and more various than might be supposed from the paper referred to. In what follows, when the mastoid cells are said to have been opened, it will be understood that the instrument used is a drill (see woodcut) made in such a manner that the distance to be penetrated is accurately regulated by a stop, so that any risk of boring too deeply is avoided. The perforation is readily accomplished in a few seconds when the incision to the bone has been made. After frequent employment of this plan I can confidently speak of its superiority both for perfect safety and for great rapidity.

In many instances, especially in the cases of adults, when it becomes a matter of immediate urgency to make an opening into the cells, and when the outer table is healthy, the advantages will be obvious to those who have had occasion to resort to this proceeding.

The following cases will illustrate the various and

serious conditions which may occur when the mastoid process becomes inflamed, and which may urgently call for the letting out of pus, the precise position of which it is not always easy to point out.

Case 1.—On January 31st, 1884, I saw, with Dr. Martin, of Somers Place, Hyde Park, a young lady, who, after an attack of acute inflammation of the left tympanum followed by a perforation of the membrane, had for some weeks continuously a most profuse purulent discharge. Beyond the very copious nature of the discharge there was no symptom which called for notice. Local treatment did not materially affect the state of the ear. There was no redness or swelling over the mastoid, and only slight tenderness on very firm and continued pressure, which was relieved by leeches and fomentations. A few weeks later, after a rise of temperature to 104° F., she had severe pain in the muscles of the left thigh, and a dragging of the foot of the same side. She walked as in infantile paralysis. The pain passed off. Early in April there were several sudden rises of temperature up to 104°, only maintained for a few hours. At no time had there been rigors. Taking into consideration the long continuance of the very profuse discharge, the symptoms connected with the thigh and impaired movements, the rises in temperature, and the fact that firm and continuous pressure on the mastoid process now for the first time gave rise to a sharp pain, although there was no œdema or redness, I advised that an opening should be drilled into the mastoid cells. Sir James Paget saw this case independently of me, and agreed as to the advisability of this proceeding. When the incision was made the bone was healthy. In spite of this the bone was drilled, and on removing the drill fœtid matter escaped through the wound. No further bad symptom impeded recovery.

Case 2.—On May 30th, 1883, a gentleman, æt. 35, gave me this history. Since the middle of the previous January, when he had acute pain in the left ear followed by

some purulent discharge, he had almost constant pain over the mastoid region. There was a perforation of the tympanic membrane, and some, though not profuse, discharge. There was considerable tenderness and redness over the mastoid process. On his return to Lincolnshire that day he applied two leeches, and afterwards hot fomentations, which gave him for a few days great relief. However, he soon began to suffer as before, and did not come to town again until October. There was then a good deal of swelling, the redness was much increased, and there was considerable œdema.

On cutting down on to the bone there was a large area quite exposed, but no pus was found in the mastoid cells. A few days afterwards a long probe was passed from the wound in a downward direction along the border of the sterno-mastoid muscle to the lower border of the thyroid cartilage. Here I made a second opening and passed a drainage-tube through. At a later date, a third opening was made down to the bone about two inches behind the first one, letting out more pus. The pain now shifted further back, and pressure over the splenius capitis caused pus to pour out of the external canal of the ear.

Sir James Paget now saw the patient with me, and decided that another exit must be made for pus in this part of the occipital region.

After a troublesome dissection in this situation matter was found in contact with the bone. Thus pus had burrowed under the scalp in the following direction. From the tympanum through the bone to a point outside the mastoid process, thence to another point under the splenius and complexus muscles. Between the last opening and the second a drainage-tube was passed. A very large area of the bone was found exposed. The patient made a good recovery.

Case 3.—A case in some respects like the first narrated occurred in St. George's Hospital in July, 1884. A middle-aged woman, after an attack of acute inflammation of the

right tympanum followed by rise of temperature, severe rigors, and local evidence of matter within the mastoid cells, came under my care. On making the incision the bone was perfectly healthy, but on perforating the cells a quantity of fœtid matter escaped. All urgent symptoms passed away at once. A week afterwards, beyond a slight discharge from the wound, the patient had no inconvenience. She was, however, seized with a rigor: this was followed by pus in the knee-joint; the joint was opened, but pus spreading upwards and the joint becoming disorganised, it became necessary to amputate the limb above the knee. This was done by Mr. Haward, and the patient made a good recovery.

Case 4.—A boy whom I saw for the first time on December 6th, 1883, had a profuse discharge from a recent perforation in the left tympanum. There was pain and swelling over the mastoid process extending above the ear into the temporal region. Free vertical incisions were made through the scalp over the mastoid, and on the left temple, letting out pus. The bone was denuded, and a probe could be passed under the scalp from one opening to the other. A few days later, further swelling about three inches behind and above the first opening made it necessary to make another incision in the occipital region. The forefinger, passed into all three incisions, showed that very large areas of the bone had been exposed. Notwithstanding the free opening, pain continued with great severity and was accompanied with great constitutional disturbance.

A general cellulitis of the scalp now set in and a fatal ending seemed almost certain. Sir James Paget now saw the case and advised further enlargement of the wounds, and agreed as to the necessity of further search for matter. The openings were enlarged. The boy's condition slowly improved, and when able to travel he was taken abroad. I saw him in October of this year, and was told that the opening over the mastoid had only recently completely healed.

Case 5.—On three or four occasions, at long intervals extending over three years, I had seen a young man in consequence of occasional head pains and giddiness depending on a perforation of the left tympanum, in which diseased bone was evidently present.

At the beginning of 1884, whilst he was a few miles from home, he was seized with acute pain in the head and intense giddiness. He was taken home, and in the course of a few hours became delirious and afterwards partially unconscious. For two or three days previously he had complained of a feeling of fulness, and of some pain over the mastoid bone. On the next day, when I saw him, he was in the same state. The sclerotics were much injected.

Although there was no swelling, but some redness, and no œdema, I thought that under the circumstances it would be best to perforate the mastoid cells with a drill. At the time he was so far unconscious that he recognised no one, nor subsequently did he ever remember what had occurred. The mastoid cells were so gorged with blood that a large quantity poured out when I removed the drill. There was no pus in the cells. Within two hours of the operation he became perfectly conscious. This young man suffered for many weeks from various symptoms of cerebral irritation, occasional extreme intolerance of light, severe headaches being the most prominent. It must be admitted that had I not formed an opinion (proved to be erroneous) as to the probable presence of pus within the mastoid cells I should not have proceeded as I did. The error in judgment, however, was most likely of material good as regards the loss of blood.

Case 6.—In referring to one more case, I beg to call attention to the occasionally slow and insidious course of the morbid process which may eventuate in pus within the mastoid cells.

A gentleman between 50 and 60 years of age, first consulted me on Feb. 11th, 1884, for a catarrh of the

tympanum which had come on after a severe cold on January 29th, and was accompanied with a feeling of numbness and weight in the mastoid region. I saw him at intervals of a month, or a fortnight, up to the end of July, during which time he had occasional leeching and counter-irritation over the process. At no time was the pain severe enough to interfere with the daily duties of a very active political life, and at no time was there more than slight pain on very firm pressure over the bone. At the end of July, in anticipation of the probable necessity of perforating the cells at a later date, he was seen by Sir James Paget in consultation with myself. There were then no urgent symptoms beyond very slight œdema on firm pressure. It was therefore decided to wait for clearer symptoms of pus within the bone. I did not again see him till October 11th, when he told me that three days previously there had been very decided pain. On the next day I let out a quantity of fœtid pus from the cells, the outer table of bone being quite softened by caries. Throughout the entire illness he had no constitutional disturbance.

I am encouraged to bring these cases before the Society by the belief that perforation of the mastoid cells is more frequently and urgently called for than might be supposed from the literature of the subject.

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 328.)

# CASE

OF

# DOUBLE SIMULTANEOUS DISTAL LIGATURE

FOR

# INNOMINATE ANEURYSM.

BY

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Received July 14th, 1884.—Read January 27th, 1885.

Laura H—, æt. 48, married, was admitted under my care into Charing Cross Hospital, Golding Ward, 16th February, 1884.

Both parents died when patient was too young to know of their diseases. She has had two sisters; one died of "cholera," the other is healthy and strong. Her relatives on the mother's side have suffered from heart affections. Patient's health has until lately been uninterruptedly good. She has had six children born living and healthy; two born dead at full term; four miscarriages.

Eighteen months previous to her admission she fell forwards upon her chest on the floor of an omnibus. No

bruising or other immediate ill-effects were noticed. A month after this she spat up one morning about a table-spoonful of inspissated blood, but had at that time no further trouble.

A year ago, that is six months after the accident, she noticed an uncomfortable sense of throbbing on the right side, below the collar-bone; soon after a pain about the elbow, which shortly extended up the arm to the shoulder and became very severe; movement and power of the arm rapidly diminished. Two months ago she had what she terms rheumatic fever; it was confined to the upper extremity, particularly to the hands, especially of the right side.

*State on admission.*—Patient looks older than her stated years; is pale, thin, and anæmic, has a dry skin and a temperature of 102·6°. She has some dyspnœa (very shallow breathing with evident effort), also a dry brassy cough, considerable loss of vocal power, the voice being toneless and low.[1]

The veins of the upper part of the chest are full and congested, so also are those of the arms, equally so on both sides. The right half of the sternum, the inner part of the clavicle, and the two upper costal cartilages of the same side with their interspaces are involved in a distinct fulness or protrusion, circular in form, and a little more than three inches in diameter, and over all this space pulsation is distinct both to sight and touch, the throb is most evident in a semicircle below and outside the sterno-clavicular joint; the clavicle and the two upper costal cartilages participate in the pulsation. In the outer half of the episternal notch, and also behind the inner part of the sterno-mastoid muscle, a pulsatile tumour can be felt; its outer border reaches as far as, but not beyond, the interspace between the two heads of the muscle. When the sterno-mastoid and the fasciæ in front of the neck are relaxed by bending the head forward, deeper palpa-

[1] This condition has come on, she says, only during the last week. Some of her friends noticed it earlier.

tion shows that the tumour, merging out of the chest, does not merely cover but actually involves the carotid.

All that part of the chest, which I have described as protruding, is absolutely dull on percussion, and relative dulness extends beyond the limit of tumefaction and of pulsation. The dull area is separated from the cardiac dulness by a line of resonance. Only a small quantity of air enters the lungs, with râles and blowing murmurs throughout; in places also, markedly at the left base, there is bronchophony. The left posterior base is more especially dull, and less air enters that part of the lung than elsewhere; here, too, the râles are most marked. The apex-beat of the heart is not displaced; the organ is healthy, but the second sound is exaggerated; this exaggeration is very distinctly marked over the site of the tumour and to a less degree over a great part of the chest. Patient has much palpitation and præcordial pain even on slight exertion. The right radial pulse is very small and faint, the left one is regular and full, 96. The pulse of the right temporal artery is markedly larger and fuller than that of the left, also the beat of the right carotid on a level with the cricoid cartilage is stronger and bigger.

She keeps the right arm very motionless and close to her side; the elbow is semiflexed. The limb is markedly atrophied; the muscles are flabby and flaccid.

| | Right. | Left. |
|---|---|---|
| Middle of upper arm . . | 8½ in. | 9½ in. |
| Forearm three inches below elbow | 8¼ in. | 9 in. |

Extension of the elbow, abduction, and rotation of the shoulder produce considerable pain, but movement of the wrist and fingers is painless. The power of the grasp is diminished; tested by the dynamometer it stands as 6½ right against 12 left.

There is a good deal of neurotic pain in the arm, running up in the course of the nerves with formication and numbness of the hands and fingers.

The patient's appetite is very bad. Tongue rather

brown but moist; some thirst. She complains of a severe pain on swallowing, which she refers to a spot on a level with the sixth cervical vertebra, about three inches to the right of the spine. Although she has pain in various parts, as in the arm and chest, this appears to be the most severe and least bearable of her troubles. On applying the stethoscope to the last cervical vertebræ while she swallows the act is heard to be prolonged, only occasionally reduplicated.

The retina of the right eye presented nothing abnormal. The vocal cords looked a little relaxed, equally so on both sides.

During the twelve days ensuing she was carefully watched and frequently examined. She had during this time a good deal of pain in the abdomen and some diarrhœa. She appeared to be losing strength, while the lungs were becoming more impervious, less air entering them, the dulness of the bases extending higher. Her voice became more and more feeble. Her temperature was often below the normal, notably on the 19th and 20th it was 97°; on the 21st 97·4°.

The most noteworthy phenomenon, however, was entire cessation of the right radial pulse—this occurred on the 20th—four days after her admission. The pulselessness extended throughout the brachial and all accessible parts of the axillary, also, I believe, to the subclavian, the third part of which could not be felt to beat.

The symptoms just described clearly indicated that I had in this case to do with a high innominate aneurysm, but certain occurrences are worthy of remark, namely, the very feeble state of the right radial pulse on her first admission and its subsequent total extinction. If together with these symptoms we collate the fact that almost simultaneously with the first sense of throbbing, that is to say, almost simultaneously with the first sign of disease —severe pain in the arm was felt, we can only come to one conclusion, namely, that the aneurysm which affected the upper part of the innominate and its offshoots had at

first inclined chiefly to the right and had more especially involved the subclavian branch; and that afterwards this part was obliterated, while remaining empty or nearly so, by compression of the carotid portion of the tumour. It is of course also possible that the obliteration was caused by detachment and subsequent impaction of a piece of clot. On this point I would not dogmatise; but must point out that spontaneous cure by impaction of clot leads to solidification with the sac full; and one should under such circumstances be able to feel a tumour behind the clavicular part of the sterno-mastoid muscle. Such tumour was entirely absent in this case.

Compression of an artery by an aneurysm situated upon it is by no means an unheard-of occurrence, though by some authors its possibility has been doubted.[1]

In this case the mechanism of such compression would be as follows:—I have elsewhere pointed out[2] that the usual idea of the mode in which the innominate bifurcates is incorrect; it is described and depicted as though the two branches arose side by side and almost at right angles to each other. In reality the subclavian springs from its parent trunk almost directly behind the carotid and runs up some distance close, almost parallel and posterior to that vessel. If this position of parts be borne in mind, there will be no difficulty in perceiving how an aneurysm involving the root of the carotid would very easily compress against the vertebræ, the commencement of the subclavian, more especially if that branch were also aneurysmal. It will also be seen how under such circumstances that artery and its dilatation would be empty or nearly empty.

The aphonia noted in this case was due to compression

[1] This idea was due originally to Sir E. Home; see Astley Cooper, 'Med.-Chir. Trans.,' vol. i, p. 12. I watched a case of aorto-innominate aneurysm, in which the large sac, curling over and compressing distal parts of the subclavian, induced considerable consolidation of the outer part of the tumour.

[2] 'Encyclopædia of Surgery,' vol. iii, p. 514.

of the trachea as also was the condition of the lungs. This latter was a most interesting example of the mode of disintegration caused by such pressure. It is produced, as the late Dr. Pearson Irvine[1] and I showed, by obstruction, not to inspiration, but to expectoration, whence accumulation of secreta, &c., in the larger then in the smaller bronchi and air-cells, necessarily inducing consolidation, parenchymatous pneumonia, and breaking down of lung tissue. The first part of this process I had in this case the opportunity of watching from day to day.

Having watched the case for ten days, waiting to see if such process has had occurred in the subclavian would extend to other parts of the sac, I came to the conclusion that the advancing morbid condition of the lungs was the principal cause of the patient's debility and the chief source of her danger. Indeed, although anæmia is not a condition favorable to the success of deligation, I foresaw clearly that postponement would still more jeopardise the lungs and would render their condition fatal, if even some weeks hence the aneurysm could be cured. Still, before proposing any operation it was necessary, since the subclavian artery was occluded, to ascertain whether the cerebral functions would be carried on without the supply of blood derived from the right carotid. I therefore compressed this vessel with my thumb; no result followed. Simultaneous compression of both carotids produced, in about five seconds, pallor of the face, a sense of swimming in the head, and a distinct depression in the power of the pulse. The two first symptoms were doubtless due to loss of circulation through the carotids, the last to interference with the functions of the vagus nerve.

Having, however, proved that occlusion of the right carotid produced no cerebral disturbance, I determined first to tie that vessel and to be guided by the immediate result as to any further action.

February 28th.—I tied the common carotid artery with

[1] 'Pathological Transactions,' vol. xxviii, p. 67, and my book on 'Aneurysm, especially of the Thorax and Root of the Neck,' p. 101.

an ox aorta ligature about one inch below its point of bifurcation. I begged that the right pupil might be watched at the moment of tightening the ligature. One observer thought he perceived a very slight contraction of the pupil, the other believed that no change took place.

After this deligation the clavicle and the protruded part of the chest pulsated more violently than before. I tried to make out if the third part of the subclavian pulsated, but the powerful throb of the clavicle rendered it impossible to ascertain this point. Fearing, however, that occlusion of the stream in the carotid might divert blood into the subclavian and thus reopen that part of the aneurysm, my colleagues also, on rapid consultation, coming to the same conclusion, I laid bare the third part of the subclavian. It was empty, and there was not the slightest pulsation in it, nevertheless having exposed it, I, with a view to future possibilities, tied it.

In the evening of the same day the pulsation had very much diminished. On the next day she began to expectorate the muco-pus that had for some time past been accumulating in her lungs. On the fourth day she lost the pain on swallowing, the voice began to improve and the protrusion of the upper part of the right chest to flatten.

March 20th.—All these signs of diminution in the bulk of the aneurysm continued to increase. On the twenty-first day she sat up and was pleased to find that she could painlessly feed herself with the right hand.

28th.—The cervical part of the tumour had apparently become nearly solid; the pulsation that remained had in great degree lost its expansile quality and had assumed more nearly the character of a communicated heave from the aorta. But at this date that enlargement appeared to increase, and although she was kept very still and quiet the hitherto solidifying tumour became softer, the pulsation resumed its expansile character, and although the thoracic protuberance did not at the same time increase, the clavicle again began to pulsate. The patient averred

that she again felt the throbbing, but none other of the subjective symptoms recurred.

April 4th.—The cervical portion of the aneurysm had become almost as large and nearly as pulsatile as it was before the operation, the thoracic portion continuing meanwhile to become flatter, and the resonance of this part to increase. With a bandage of elastic webbing I bound upon the tumour a pad of cotton-wool, for which in two days I substituted a round, hollow, elastic ball, according to a method successfully employed by Mr. Holmes in a case of subclavian aneurysm.[1] This treatment produced a good deal of pain and considerable restlessness, therefore, as at the end of a week no benefit had resulted, it was discontinued. I was considering the desirability of employing needles, with or without the galvanic current, when on the 15th the tumour was found to be smaller and more solid.

20th.—Solidification and decrease in size of the cervical portion of the tumour has been progressing regularly since the last report. To-day she was allowed to get up.

May 6th.—The cervical part of the aneurysm appears as a small solid tumour behind the inner head of the sterno-mastoid muscle; it is lifted by each beat of the aorta, but has no inherent pulsation. The voice is natural, she has no cough, suffers, however, a good deal from muscular rheumatism, and is feeble. She left the hospital desirous of remaining at home for some time before going to a convalescent institution.

June 4th.—The patient showed herself at the hospital. There is no protrusion of the right upper half of the chest nor any tumour to be felt in the neck. Over the late site of the aneurysm percussion notes all clear, and respiratory murmurs are to be heard where previously only exaggerated heart-sounds were audible. The lungs are clear down to their lowest bases.

No pulse could be felt in any branch of the right carotid, nor of the subclavian. The right arm, which has

[1] 'Lancet,' Feb. 12th, 1876.

hitherto been always rather cooler than the left, is now rather warmer, but the temperature of the face is lower on the right side. It is hoped that the patient will attend. She was last seen at the end of August, well, as far as aneurysm is concerned.

*Remarks.*—As I have already made some remarks concerning the diagnosis of this case, the few observations I would offer now shall be very brief.

The disease was distinctly an example of high innominate aneurysm, eminently fitted as far as situation is concerned for the double distal ligature.

It is possible that some of the Fellows may see a different mode from that above given of accounting for the singular occlusion, by the disease itself, of the subclavian artery and its branches. Yet I would point out that whatever view be taken of the occurrence it cannot be supposed to have taken place elsewhere, than at or near the commencement of the vessel, and proximal to the vertebral artery. With this fact in view, I tested, as above detailed, the effect of temporarily cutting off the blood-stream in the carotids. After deligation of the right common carotid artery no cerebral symptoms whatever supervened, although in all probability no blood found its way directly to the right side of the brain. I hold this fact to be highly important as aiding to prove a view, elsewhere expressed,[1] that occlusion of a carotid by ligature or otherwise has no injurious effect upon a sound and healthy brain.

Whether or not the subclavian artery should also be tied remained in doubt until after the operation on the carotid; I was influenced at that time by the increased pulsation of the aneurysm, and believe that I may congratulate myself on the course taken.

The lungs were, when I operated, becoming rapidly filled up with retained secretions. The cessation of pressure on the trachea had almost immediately the effect of permitting the elimination of large quantities of muco-

[1] 'On Aneurysm, especially of the Thorax and Root of the Neck,' p. 81.

pus, and subsequently of mucus, the lungs becoming by that process disburdened. To watch the gradual disappearance of the pulmonary symptoms, to hear the dulness clear away and the râles diminish, was a most interesting clinical experience.

One other point I would like to touch upon. The tumour, or rather that portion of it which was in the neck, had become by the 20th March (twenty-first day of operation) nearly solid, was tolerably hard, and had considerably diminished; about a week later it again began to soften and to increase until, though the thoracic portion did not yield in the same way, the cervical part was nearly as large as before the operation, the blood in it being evidently fluid. As is well known, recurrence of pulsation occasionally occurs after a certain consolidation of the aneurysm has followed treatment either by the elastic band, by pressure, or ligature, though I am not aware that ultimate success has previously been observed to follow such recurrence in the neck. But the questions which I would put to the Society are these:

What becomes of the clot (probably soft clot) already formed in the sac, and which quite disappears when the recurrent pulsation persists a certain time?

Since no sign of embolism or plugging of vessels accompanies these phenomena, it would appear that such clot must again become fluid. If so, what peculiar power has living and flowing blood that can enable it to dissolve and liquefy clots formed from its own substance?

The answers to these questions involve, I believe, some important points in physiology and in pathology.

It may be well to remind the Fellows of the Society that since I last had the honour of addressing it on this subject, two cases of aneurysm about the upper part of the thorax and the root of the neck have been laid before it, the one by Mr. Howard Marsh—he tied both vessels simultaneously but without success; another by Mr. Morris, who attempted, but failed, in the altered condition of the parts to tie the carotid—this patient also died:

I have to thank the Hon. James George Beaney, of Victoria, for sending me a copy of his 'Clinical Lectures.' One of them gives two cases of innominate aneurysm treated by consecutive ligature of the carotid and subclavian (third part); they were both eminently successful. In neither did the carotid ligature produce any marked benefit until the subclavian was tied, four and two weeks afterwards respectively.

I may be permitted also to recall the fact that this is the sixth case of simultaneous double distal ligature that I have reported to this Society. Of these, one was for a very large aorto-innominate aneurysm (*a*), two for smaller but still considerable aneurysms of the same description (*b*, *c*), two (including this case) for aneurysms purely innominate (*d*, *e*), one for aneurysm of the first part of the aorta (*f*).

The first was one of my earliest cases, such as with my present increased knowledge I should not subject to any operation. The other five have been all successful.

*a*. J. L—, from Bath; operated 6th December, 1877.
*b*. Robert W—; operated 14th August, 1877.
*c*. Laura G—; operated 6th December, 1877.
*d*. Catherine H—; 7th January, 1878.
*e*. Laura H— (this case); 28th March, 1884.
*f*. John S—; 15th February, 1879.

*Note.*—It was reported to me in May, 1885, that the patient was quite well, and going about her household duties with ease and comfort.

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 332.)

# CASE

OF

# DISPLACEMENT AND FRACTURE OF THE AXIS.

## LIFE PROLONGED FOR TEN YEARS.

BY

D. LOWSON, M.D.,
HULL.

(COMMUNICATED BY SIR PRESCOTT HEWETT, BART., F.R.S.)

(Received November 11th, 1884—Read February 10th, 1885.)

DISLOCATION, or fracture of the spine, if situated above the origin of the phrenic, is as a rule immediately fatal. A few exceptions have occurred. One of these, which I have kept under observation for ten years, is unique in manifesting no paralysis nor other symptoms of nerve injury. The following is the history of the case:—

On a dark night in the winter of 1874 W.P—, an old man of 75, was walking along the highway that runs between Wakefield and Huddersfield. Coming to a sudden bend in the road, which in the darkness he did not observe, he held straight on and fell headlong over a sunk wall six feet down into the field below, where for some time he lay stunned and senseless. On recovering consciousness he experienced excruciating pain in the neck. He made the

best of his way home to his cottage, where he lived all by himself, undressed and went to bed without any assistance and slept throughout the whole night. Next morning the pain had moderated, but he could not raise his head without a return of the agony. He managed to get out of bed by pushing his feet out first and lifting his head with both hands. The same day he consulted a neighbouring surgeon, who ordered him a liniment, but apparently did not recognise the nature of the injury. A fortnight after this he became an out-patient at the Huddersfield Infirmary, and it was here I first saw him. At first sight I took it to be a muscular or ligamentous sprain, but the history and the man's nasal intonation in articulating led me to examine him more minutely.

He carried his head very carefully and rotation was not impossible, though limited and painful. The chin was tilted upwards a little, the occiput depressed as if seeking for support from the collar of his coat, and the neck was bent backwards. The mouth was kept open, the respiration being mainly oral, while the nasal breathing was obstructed. Deglutition of solids was difficult, but liquids were swallowed without much trouble. On examining the throat, the palate and uvula were seen to be pushed forward by a tumour projecting from the posterior wall of the pharynx. This tumour was hard and was covered by healthy mucous membrane, which at the most prominent point was pale and anæmic from tension. The apex of the swelling resembled the projecting lip of a cervical vertebra. There was no paralysis and no anæsthetic or paræsthetic symptoms. From the position of the prominent part I thought it was the third vertebra, which I believed had sprung forward from the force of the concussion, there being at the same time probably bilateral fracture of the neural arch. An attempt was made to give artificial support to the injured spine, but the patient preferred to be without it. He left the hospital after being an in-patient for three weeks.

Three years after the patient returned to have a

glaucomatous eye removed. At this time many of the symptoms had improved. There was no pain. Rotation of the head was much more extensive, and deglutition was easy. He died in July of this year, 1884, from senile gangrene. A post-mortem examination was obtained with some difficulty, and the cervical spine was extracted and carried away. After removal of the soft parts the following state of matters was found to exist :—

The axis with odontoid is bent back at an angle of 60° with the horizon. There is complete anchylosis between the bodies of the second and third vertebræ, with the exception of a small angular interval in front which is filled with the remnant of the intervertebral cartilage. The arch of the axis overlaps and encloses that of the third and is anchylosed to it. The upper articular process of the third is merged in the pedicle of the axis. The lower articular process of the axis is widely separated from the transverse process and is situated on a level with and behind the same process of the third, from which it is separated by a narrow groove. The two bones now form one mass. As regards the atlas there are two anatomical anomalies viz :—(1) A foramen behind the articular process instead of the usual groove for the vertebral artery. (2) A bony process projecting from the posterior arch to rest on the root of the spinous process.

In accounting for the mode in which the injuries have occurred it seems probable that, in falling, the vertex came first in contact with the ground, and the impetus of the trunk impacted the upper articular process of the third cervical vertebra into the pedicle of the axis. (The probability of a fracture in this locality is increased by the great separation of the transverse and lower articular processes.) The trunk then falling backwards over the head, doubled up the neck, burst or stretched the anterior common ligament, and drove forward the lower part of the body of the axis ; the apex of the odontoid, the atlas and skull were carried back, and the arch of the third was telescoped into that of the second.

The diagnosis was made by the prominence in the pharynx. The history of the fall, and the presence of obstructive symptoms to deglutition and respiration, which did not exist before the injury, excluded the idea of a bony outgrowth. The pain and inability to raise the head were also confirmatory.

Two cases similar to the above were for some time under the care of Sir Prescott Hewett. The symptoms of both were nearly identical, but differed from the case related in having partial paralysis which ultimately passed away. Tumours in the pharynx existed in these. The head was bent back and there was great pain. One of these is still alive, the other has been lost sight of.

An example of a like injury was picked up in a churchyard and has been described in the 'Medico-Chirurgical Transactions' by Sir James Paget.[1] Professor Flower describes in the 'Transactions of the Zoological Society' an injury to the neck of a whale which I believe is not unlike those already mentioned. The skeleton of the animal is in the College of Surgeons' Museum.

[1] 'Med.-Chir. Trans.,' vol. xxxi, p. 285, and vol. xxxii, p. 177.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 336.)

# OBSERVATIONS

ON THE

# RADICAL CURE OF CLUB-FOOT,

## AND EXHIBITION OF CASES WHICH HAVE BEEN OPERATED ON.

BY

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Received August 28th, 1884—Read February 10th, 1885.

As much thought has been bestowed within the last decade on the radical cure of confirmed club-foot, it may not be devoid of interest to recapitulate some of the leading facts gained by experience, and to submit conclusions thereon to the judgment of this Society.

I will divide the consideration of the subject under the following headings :—

1. Some reasons that have suggested operative procedures in cases of intractable club-foot.

2. The cases especially suited for operation.

3. The operation itself, the description of its details, and the necessary instruments.

4. The list of operations (26) performed by myself; and notes of other cases operated on by British and foreign surgeons.

5. The exhibition of casts taken before and after operation, the osseous wedges removed, and living specimens of the results gained.

6. Concluding remarks.

1. Some reasons that have suggested operative procedures in cases of intractable club-foot.

The obstinate character of severe forms of club-foot, its general tendency to get worse unless subjected to continued treatment, and the discouraging relapses occurring even after proper treatment has been employed, may be mentioned as important grounds for reconsidering the usual methods of practice in vogue up till 1874. My experience showing how absolutely useless instrumentation alone was, I carefully considered the operation of removal of the cuboid bone, an operation that had been suggested by Dr. Little in 1854, and practised by Mr. Solly, of St. Thomas's Hospital, also in 1854.

From an anatomical point of view no structure of special importance is removed either in excision of the cuboid bone, or in the excision of a wedge at the transverse tarsal joint, if the operation described hereafter be practised.

2. The cases especially suited for operation.

I have never yet operated on any case that had not been previously subjected to orthopædic treatment; in some cases such treatment had been carried out during the whole of the patient's lifetime; whilst in others the condition was so incorrigible as to have necessitated the abandonment of treatment as hopeless.

3. The operation itself, the description of its details, and the necessary instruments.

The operation consists of the removal of an osseous wedge at the transverse tarsal joint; this operation has hitherto been confined to cases of talipes varus and equinus, with their combination. I will describe the operation for talipes varus first, then that for equinus.

(1) *The operation for talipes varus.*—The patient being

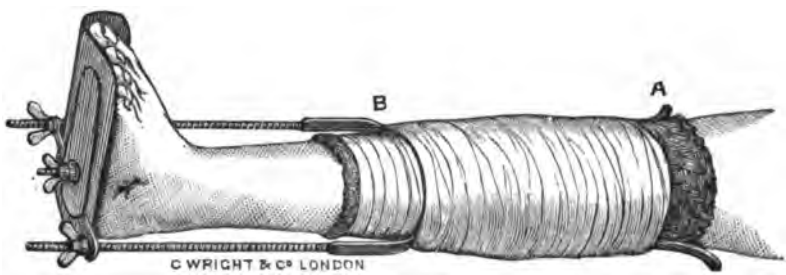
under the influence of an anæsthetic, and an elastic bandage being placed on the leg after elevation, an oblong piece of skin is excised from the outer side of the foot over the cuboid. This should include the distended and hypertrophic bursa and should correspond nearly with the size of the base of the osseous wedge required. On the inner side of the foot, at the stereotyped crease of skin, and in a line over the astragalo-scaphoid joint, a vertical cut is made of sufficient length to include the thickness of the scaphoid bone; these two imaginary lines, drawn across the dorsum of the tarsal arch, represent roughly the superficial area of the triangular piece of bone to be removed. Next, keeping close to the bones, elevate the tendons, nerves, arteries, and veins with the blunt curved knife on the dorsum, until the triangular space has been cleared from the outer to the inner side, for the transit of the kite-shaped director. Having passed the director between the tarsal bones and the soft structures above direct the probe-pointed saw successively along the grooves on the under surface of the director, and saw the wedge out with precision. It is usually better to saw the distal side of the wedge first, and the ankle-joint side last.

The blunt curved knife is next used on the plantar aspect of the wedge, the operator again keeping close to the bones, and lifting out the wedge in one piece by the aid of strong pointed bone forceps. The wedge usually embraces the component bones of the transverse tarsal joint, the cuboid bone invariably predominating; and portions of the bones anterior to the transverse tarsal joint, according to the condition of the deformity. Many of these osseous wedges are exhibited to the Society. One shows the component bones of the transverse tarsal joint alone, viz. astragalus, os calcis, scaphoid, and cuboid. Another shows portions of every one of the tarsal bones, and the bases of the four outer metatarsal bones also. (Case 25.)

By this method of operating, simple or compound wedges of bone may be removed with ease and precision;

their form can also be tested, as they each appear in the shape of one neat block instead of the *débris* of a piecemeal and haphazard dissection.

I prefer, for many reasons, to set up these compound fractures of the foot immediately that the distortion has been corrected by rotation and abduction; the sawn surfaces of the bones should be co-adapted, and maintained *in sitû* on the splint, shown in the accompanying woodcut, prior to the patient's awakening from the anæsthetic.




In applying the splint the patient's thigh, knee, and upper half of the leg must be first enveloped in a sock of thick fleecy wool (A) knitted to shape; over this are applied three or four layers of plaster-of-Paris bandage. To the surface of the latter the side irons are accurately adjusted, and are retained in position by additional turns of the plaster bandage (B). The footpiece slides over the ends of the side irons, and is regulated by a male screw, screw washer, and female thumbpiece on each side iron. Eversion of the anterior half of the foot is carried out by everting the moveable foot-plate on a pivot and fixing it by a thumb-screw (C). Any undue pressure can be at once removed; wide interruptions at the malleoli prevent any inconvenience from œdema; dressings (if thought to be desirable) may be readily applied, and the foot leaves the operating table a fixture.

I neither use sutures, nor dressings; the wounds are exposed to the air. Cleanliness, however, of a strict character is maintained, and free drainage provided for.

I have never met with hæmorrhage of a serious nature; this may be accounted for by the plan of keeping close to the bones, and bluntly dividing with the curved knife any articular or osseous branches.

(2) *The operation for talipes equinus.*—A few words will suffice to indicate the variation necessary in the operation for cases of talipes equinus.

Taking again the line of the transverse tarsal joint as a guide, on the outer and inner sides of the foot, immediately over this joint, two wedge-shaped pieces of skin are removed equal in extent to the amount of bone demanded. The soft structures are freed on the dorsum of the foot in the way previously described; but as the base of the osseous wedge for equinus cases is at the dorsum, and its apex at the sole the parallel wire director, instead of the kite-shaped varus one, is used. The saw is successively inserted in its grooves, and by keeping in mind the idea of a keystone, a clean wedge of bone is cut out from the dorsum to the sole of the foot. This wedge is easily extracted in one piece, and consists of the component bones of the transverse tarsal joint, and in severe cases of portions of bone anterior to it. The splint used is the same as for varus.

4. The list of operations (26) performed by myself, and reference to those operated on by other surgeons.

The following list embraces a summary of my own cases:—

## *Excision of Cuboid Bone and Bases of the two outer Metatarsal Bones.*

| No. | Sex. | Age. | Admission. | Operation day. | Nature of deformity. | Operation. | Discharged. | Result. |
|---|---|---|---|---|---|---|---|---|
| 1 | Boy | 15 | Jan. 25, 1874 | Jan. 27, 1874 | Talipes varus congenital, left | Excision of left cuboid | May 27, 1874 | Recovery |
| 2 | „ | 15 | „ | Mar. 17, 1874 | Talipes varus, right | Excision of right cuboid | „ | „ |
| 3 | „ | 14 | Jan. 12, 1875 | Jan. 18, 1875 | Equino-varus, right | Ditto | April 1, 1875 | „ |
| 4 | „ | 10 | Jan. 14, 1875 | Jan. 26, 1875 | Ditto | Ditto | May 26, 1875 | „ |
| 5 | „ | 10 | „ | „ | Equino-varus, left | Excision of left cuboid and fifth metatarsal base | „ | „ |

## *Excision of Osseous Wedge at the Transverse Tarsal Joint.*

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 6 | Boy | 6 | Mar. 17, 1876 | Mar. 28, 1876 | Equino-varus, left | Excision of osseous wedge at transverse tarsal joint | May 22, 1876 | Recovery |
| 7 | „ | 12 | Nov. 7, 1876 | Nov. 14, 1876 | Equino-varus, right | Ditto | May 21, 1877 | „ |
| 8 | „ | 12 | „ | Jan. 16, 1877 | Equino-varus, left | Ditto | „ | „ |
| 9 | Man | 20 | Nov. 6, 1877 | Nov. 20, 1877 | Talipes equinus, left | Dorsal wedge excised | Dec. 5, 1877 | D. in 14 d. |
| 10 | Girl | 1½ | Feb. 22, 1878 | Mar. 5, 1878 | Talipes varus, right | Excision of osseous wedge at transverse tarsal joint. | April 12, 1878 | Recovery |
| 11 | Boy | 16 | May 20, 1878 | May 31, 1878 | Equino-varus congenital, right | Excision of wedge at transverse tarsal joint | Aug. 29, 1878 | „ |
| 12 | „ | 14 | May 8, 1878 | May 25, 1878 | Equino-varus, left | | Aug. 31, 1878 | „ |
| 13 | „ | 10 | July 6, 1879 | July 8, 1879 | Talipes varus, right | Ditto | Sept. 9, 1879 | „ |
| 14 | „ | 4 | June 6, 1881 | June 7, 1881 | Talipes varus, left | Ditto | July 22, 1881 | „ |
| 15 | „ | 10 | June 10, 1881 | June 14, 1881 | Paralytic equino-varus, right | Ditto | Aug. 9, 1881 | „ |
| 16 | „ | 10 | June 22, 1881 | June 25, 1881 | Talipes equinus, left | Ditto | Aug. 11, 1881 | „ |
| 17 | „ | 11 | July 28, 1881 | Aug. 4, 1881 | Equino-varus, right | Ditto | Oct. 21, 1881 | „ |
| 18 | „ | 5 | Nov. 25, 1881 | Nov. 29, 1881 | Talipes varus, right | Ditto | Feb. 13, 1881 | „ |
| 19 | Girl | 4 | Mar. 27, 1882 | Mar. 31, 1882 | Congenital varus, right; malformation developed, left foot | Ditto | June 1, 1882 | „ |
| 20 | „ | 13 | June 1, 1882 | June 6, 1882 | Equino-varus, left | Ditto | Sept. 9, 1882 | „ |
| 21 | Boy | 3½ | June 17, 1882 | June 20, 1882 | Double congenital talipes varus | Both trans. tars. joints excised | Aug. 21, 1882 | „ |
| 22 | „ | 10 | June 25, 1883 | June 26, 1883 | Cong. talipes equino-varus, left | Excision of wedge at left transverse tarsal joint | Dec. 19, 1883 | „ |
| 23 | „ | 7 | July 9, 1883 | July 10, 1883 | Cong. double talipes equino-varus, especially right | Excision of right transverse tarsal joint | Oct. 19, 1883 | „ |
| 24 | Girl | 4 | Nov. 13, 1883 | Nov. 20, 1883 | Ditto | Ditto | Mar. 21, 1884 | „ |
| 25 | Boy | 18 | Jan. 8, 1884 | Jan. 11, 1884 | Cong. left talipes equino-varus, toes upside down | Rectangular wedge excised at transverse tarsal joint | Mar. 21, 1884 | „ |

*Note.*—In 1885 I have successfully operated on two more males.

Up to the present 23 individuals have furnished 26 distinct operations. Two have submitted to the double operation at one sitting, 19 are boys, 4 are girls. Fourteen are cases of talipes equino-varus, ten are cases of varus, two cases of talipes equinus. My oldest case is 20, the youngest 1 year and 4 months. I regret to state that I have lost one out of the 26 operations, giving a mortality percentage of $3\frac{11}{13}$.

The shortest stay in the hospital occurs in Case 10 (38 days); the longest stay is 175 days, in Case 22; this prolonged residence was due to domestic causes. The average stay in hospital, from the day of operation to the day of discharge, is 77 days.

All the patients, save the one whose case ended fatally, have been enabled to walk, and perform the daily routine of work, subsequent to the operation. In some cases, the application of a plaster-of-Paris or gum and chalk bandage has been found necessary for a time, as in the case of other resections. A high boot must be worn by others. Patients, after the operation, become absolutely plantigrade, the scar is small and well out of the line of pressure; relapses are prevented, and a useful though shortened foot results.

In recording the work done by British and foreign surgeons in similar cases, the list drawn up by Dr. Granville Faught for Dr. De Forest Willard,[1] of the University of Pennsylvania, is the most complete. Tables are given of excisions of the cuboid bone, of the astragalus, and of wedge-shaped excisions at the transverse tarsal joint.

Amongst British surgeons the pioneer was the late Mr. Solly, who removed the cuboid in 1854;[2] Mr. Davies-Colley[3] also performed a similar operation in 1875.

5. The exhibition of casts taken before and after operation, the osseous wedges removed, and living specimens of the results gained.

[1] 'Club-foot,' by Dr. De Forest Willard, Philadelphia.

[2] 'Medico-Chirurgical Transactions,' vol. xl (1857), p. 118.

[3] Ibid., vol. lx (1877), p. 11.

On the table are thirty casts illustrating especially the condition of the feet before and after operation, specimens are also presented showing how the osseous wedge is excised in one block; in all varus cases the cuboid bone predominates.

Specimen No. 25 includes part of all the bones of the tarsus, and the bases of the outer four metatarsal bones. It is seen that the head of the astragalus is dwindled and misshapen, reminding the observer more of the concavo-convex aspect of the trapezium. The scapho-cuboid articulation is exceedingly pronounced, the bones generally are not well developed, the ligaments are tough and hypertrophied, a small bursa is to be seen over the anterior outer aspect of the os calcis.

Seven living specimens are exhibited. One a young man who was operated on seven years ago, and who has never worn any special instrument since. Two of the cases are wearing a high cork sole.

6. General observations on club-foot, with conclusions.

After an excision at the transverse tarsal joint the ankle-joint (as a rule) remains unimpaired; but I have seen stiffening and loss of free motion result.

Excision of an osseous wedge at the transverse tarsal joint is an operation mainly reserved for inveterate cases, and for cases where milder measures have failed.

In conclusion, for cases of talipes varus, equino-varus, or equinus, excision of an osseous wedge at the transverse tarsal joint is, in my opinion, on mechanical grounds a *most* valuable operation for restoring symmetry and utility to a deformed and useless foot, no case is absolutely hopeless, or to be condemned to amputation before the surgeon has given this excision fair consideration; it will, I trust, after judicial criticism, be yet further adopted, and take its place amongst the accepted joint excisions in surgery.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 339.)

# AN ACCOUNT

OF

# TWO FAMILIES, SEVERAL MEMBERS OF WHICH ARE ATAXIC.

BY

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Some years ago the late Professor Friedreich[1] described for the first time a form of ataxia which he believed essentially to resemble the classical type of locomotor ataxy, but which differed from it in several particulars. The resemblance consists in the main symptom of incoordination in movement, beginning in the legs and spreading at a variable rate upwards; the differences are as follows: (1) Friedreich's disease attacks many members of one family, usually brothers and sisters, and females no less than males; (2) it attacks at a comparatively early period of life, sometimes quite in childhood; (3) the disorder of gait, caused by the ataxy, is uncomplicated and at first the only symptom, whereas in ordinary tabes the ataxic stage may be preceded by a large variety of symptoms,

[1] 'Virchow's Archiv,' vols. xxvi, xxvii, lxviii, lxx.

motor, sensory or trophic, and above all, by that of pain; (4) as the disease progresses, two symptoms may appear not usually seen in tabes, viz. disorder of articulation and nystagmus. These also Friedreich regarded as due to incoordination of movement. Sundry other symptoms, such as curvature of the spine and contractures of the feet, have been added. The pathology of the disease rests as yet on a few cases only; but it seems to be established that there is always systematic sclerosis of the posterior columns of the cord, and often disease of other parts of the cord as well.

I will not now go into the literature of the subject,[1] but will merely mention that the cases exhibited by Dr. Carpenter at the Medical Society (1871), Dr. Gowers at the Clinical Society ('Transactions,' vol. xiv), those published by Dr. Dreschfeld in the 'Manchester and Liverpool Hospital Reports' (1876), and by Mr. D'Arcy Power in the 'Reports of St. Bartholomew's Hospital' (1882), comprise, I believe, all that have been reported in England.

The cases which form the subject of this paper are taken from two different families; three from one, and two from another. In this second family there is a third ataxic patient whom I have not seen, and a fourth who will possibly become ataxic. None of the cases are far advanced. With respect to the upward spread of the disease, speech is affected in some only; well-marked nystagmus has not yet appeared in any.

Case 1.—Kate Woodcock, then æt. 14, was brought to me at the National Hospital for the Paralysed and Epileptic, on October 1st, 1880; the account given by her mother was that she had had measles three years previously, and soon after it a fever which the mother called rheumatic fever; after this she became subject to fits in the night-time, in which fits she screamed and became

[1] See 'Brain,' No. 25, April, 1884; and for more recent cases—(α) Schmidt's 'Jahrbuch,' December, 1884,—cases by Wälle and others. (β) 'La Rivista Clinica,' October, 1884,—cases by Musso.

insensible when she was held; next, she became unable to walk straight. She was also said to fidget with her hands and drop things. The only symptoms I could notice were that she had a twitching of the mouth and forehead just like that of chorea; and, as regards the gait, that after walking several times up and down the room she became rather unsteady. I took the case to be probably chorea, and till June, 1881, she continued to attend, but remained in much the same condition. She came again during the last three months of 1882, when she seemed, if anything, more unsteady. In January, 1883, she was laid up with scarlet fever, but came back to me in the following April. The unsteadiness had then increased so much that it was evidently due to something else than chorea. She swayed from side to side as she walked, and had difficulty in turning; she could not stand at all with her eyes shut. The patellar tendon reflex was absent on both sides. Dr. Buzzard, who was present, kindly verified this for me. The contractility of the quadriceps muscles (to faradism and to percussion) and their nutrition, were normal. Till August her condition remained stationary. It may be summarised as follows:—Gait unsteady, the left foot turns in as she walks; patellar tendon reflexes absent, plantar reflexes present, tactile sense in feet and toes normal; muscular sense normal (*i. e.* she knows the position of her limbs when her eyes are shut); occasional pain in the head, but in the limbs no pain, numbness, or paræsthesia, except a slight occasional pricking in the feet; slight awkwardness in the hands, but nothing really noticeable, speech normal, no nystagmus, pupils contracting sluggishly to light, readily for accommodation; fundus of eyes, except for some tortuosity of the retinal vessels, normal. Still some twitching of the forehead and of the left upper limb. Extensor tendons of both great toes prominent; the mother thought that the feet had become humped.

In August she became worse. She often went down on her knees in the streets, the ankles seeming to give; she

had another fit, apparently a kind of fainting fit. On August 25th I saw her at her mother's cottage, looking pale and thin, and only able to get about by holding on to the banisters and furniture; she had difficulty in directing the movements of the legs, *e.g.* in crossing and uncrossing them, or in describing a circle in the air with her foot; though she still knew how they were placed. In addition there seemed to be some actual muscular weakness and the thigh muscles were rather flabby. The Achilles tendons were becoming tight, and the feet looked short and stumpy.

She went to the Convalescent Home of the Hospital at Finchley; and since then has been at the Holborn Union Infirmary, where, by the kind permission of Dr. McLearn, I have twice seen her during the present year (1884). In March, she was in bed, unable to get about alone; there was slight general stiffness in the legs. In October, though out of bed, she could not walk; she had difficulty in managing the legs even when sitting; some awkwardness of the hands; handwriting bad, she says her head shakes when she tries to write; in reading, she separates the syllables and sometimes drops one. The legs are cold, their muscles small, some rigidity at the ankles, though none at knees; the feet more deformed than before, being in a position of equino-varus, the plantar arch high, the toes, especially the great toes, tending to turn up, and the toe-balls projecting on the plantar surface. Sensibility in feet and legs still normal, except that a prick with a needle hurts her very little.

She is a poorly-developed girl; though she is now 18, menstruation has not yet appeared.

Case 2.—John Woodcock, younger brother of the last patient, came to me first in April, 1883 (age at that time 18). The mother said that during the last two years she had noticed a "catching" in his legs, which seemed to give when he walked and to make him unsteady. This had come on gradually; she ascribed it to a fright from

an operation for removal of his tonsils. She had noticed no twitching of his face or limbs, though she said he was fidgety in his ways. Two or three months ago he had complained of pains in his limbs, apparently of an aching character. He had convulsions when a baby, measles with his sister Kate, and also the so-called rheumatic fever. When seen, he walked rather unsteadily, swaying somewhat from side to side, though not nearly so badly as his sister. With eyes shut and feet placed together he swayed about but did not fall. Patellar tendon reflexes absent (the quadriceps muscles being normal). Plantar reflexes slight, sensibility in feet normal; speech plain, no nystagmus, action of the pupils normal.

During the time of his attendance (from April to October, 1883), he did not improve. Twice he had an attack of giddiness and fell off a chair; from time to time slight irregular movements of the head were noticed; he became unable to stand with his eyes shut.

In March, 1884, I saw him with Dr. McLearn at the Infirmary; the walk was more unsteady, he lurched from side to side and, when he turned, his feet seemed to get crossed; Romberg's symptom was well marked. The nurse said he did not always know where his hands were. She had also noticed a peculiarity in his speech, not exactly a stammer, but a slowness in bringing out his words. He paused between words and syllables as he read aloud to me.

In October there was little change. I noticed that the toes had a tendency to turn up.

Case 3.—Hannah Woodcock, an elder sister of the last two patients, present age 20. In 1877, she had the so-called rheumatic fever with the others. Afterwards she used to stoop as she walked. She went to service, but her health failed. She was under Dr. Sturge at the Royal Free Hospital from October 1880, to June, 1881. She had been knocked down by a drunken woman in the street, came home all starting and trembling, and was

subject afterwards to the same "startings" and "nervousness." The mother says that she was told her daughter had the St. Vitus's dance (I mention this to show the probable similarity of onset in her own and her sister's case). Curvature of the spine was discovered, and treated at the Orthopædic Hospital, with a spinal support. This made her much better able to walk.

I have seen her on various occasions from April of last year till the present time. At first there was nothing noticeably wrong with her walking, but she has since acquired an unsteadiness of gait and occasional sideward lurch. Romberg's symptom, variable at first, is now well marked; she can neither stand with her eyes shut nor walk in the dark. Patellar tendon reflexes absent. During the present year some weakness, or rather awkwardness, of the left hand has shown itself. There is usually a droop of the left eyelid, and sometimes an internal squint of the left eye; otherwise nothing abnormal about the eyes or pupils. There is considerable lateral curvature of the spine. There is a deformity of the feet, not unlike that in her sister's case, but no well-marked equino-varus. An aching and tenderness of the feet, which she sometimes has, may be due to this deformity; over and above it I have found no sensory abnormality, nor any true paralysis of movement, nor loss of electro-contractility in the muscles. Menstruation has been regular and natural for the last two or three years.

There are four other members of this family (not including a child that died of scarlet fever); one an elder brother, age between twenty and thirty, three younger children, ages from nine to six. All these are in good health,[1] but I may note that in some of them I have been unable to obtain any patellar tendon reflex, and in others it seems to be uncertain. In the father and mother it is normal. The nervous inheritance seems to come from the mother's side. She had fits till the age of twelve, another at seventeen, and another when pregnant with

[1] Two have since shown symptoms of disease. See note on p. 157.

the first of these patients (Kate). She has no brothers and but one sister, living at Hailsham. This sister is insane, but has nevertheless a large family. This family seemed to me quite healthy, but I had difficulty in obtaining tendon reflexes in the two youngest members.

As to the maternal grandfather of my patients, there seems good reason for thinking he was ataxic. He was taken ill at the age of fifty, had severe pains in his legs, for which he had to take opium; walked with unsteadiness as if he were drunk; finally took to crutches, and died at the age of seventy-five of bronchitis, having had two or three fits.

In the next family there are three cases of ataxy, and one girl who will probably become ataxic.

Case 4.—Ruth Harriet Harmer, æt. 20. I have seen her at her mother's cottage on two or three occasions. Her condition is as follows: — She sits in a humped-up position, but there is, I am told, no spinal curvature; her head sometimes sways a little. When she stands there are constant balancing movements of the body, and she becomes more unsteady when she shuts her eyes. She can only walk by supporting herself against the wall and furniture, and seems as if she would tumble to one side. The patellar tendon reflexes are absent. There is, I think, some actual muscular weakness of the lower limbs, but they are well nourished and the electro-contractility of the muscles is normal. When she tries to describe a circle with her foot she makes a very irregular figure. The cutaneous sensibility of the feet, in all its modes, and the muscular sense are normal. The skin of the feet and legs is cold and mottled. The plantar arch is high and the foot stumpy looking. Her speech sometimes appears to be slow and drawling; her mother says it becomes thick if she reads for long. There is a certain tremulousness of the eyeballs as she follows an object, but no definite nystagmus. The pupils act normally to light. The upper limbs are but slightly affected, if at all.

The unsteadiness has been noticed ever since she was thirteen, but she has been bad for two or three years, since an attack of "low fever." (Her mother and another sister had this same fever; the symptoms were sorethroat, bad cold, and feverishness, with much debility afterwards.) In October, 1882, she became subject to cramp in the limbs if they were long in one position. A year later sharp shooting pains in the legs began, and tearing pains in the loins. These improved during the recent hot summer. Her menses began at seventeen, and have been regular and natural since.

Case 5.—Alice Rose Harmer, æt. 16, sister of the last patient. As in her sister's case it was noticed that from the age of thirteen she was apt to fall and tumble about, but her walking has been worse the last two or three years. She now walks unsteadily, and occasionally reels from side to side; she is slightly unsteady when she stands with her eyes shut. Patellar tendon reflexes absent; muscular power unimpaired; farado-contractility of muscles normal. Sensory functions normal in every way. No pain. There is the same chilliness and mottling of the legs as in her sister's case, and the same shape of foot. She has had during the present year a small but rather deep ulcer on each middle finger. No marked ataxy of upper limbs. Speech not noticeably affected. No nystagmus. She is pallid, and subject to severe headaches, which often end with vomiting. Menses regular since the age of fourteen or fifteen; twice during the present year they have been profuse and accompanied with epistaxis.

There is an elder brother (æt. 21) in New Zealand; since he went there he has become hump-backed and unable to walk about; he, like his sisters, used to fall about when he tried to run from the age of thirteen onwards.

A third sister, æt. 11 or 12, has been brought to me

because the mother thinks she is beginning to walk like the other two girls. I cannot corroborate this myself, but the patellar tendon reflexes are absent, or nearly so.

There are five others, all said to be healthy. Two have died of convulsions and there have been two stillborn. The father appears to be not strictly sober; but neither in the parents nor grandparents can I make out any definite history of nervous disease.

The chief interest in this class of cases attaches to the ætiology. There is a family predisposition, not merely in the sense of a neurotic diathesis manifesting itself in varions forms, such as epilepsy, insanity, migraine, &c., but predisposition to organic disease of a particular part, viz. sclerosis of the posterior columns, with possibly other parts of the cord. The repetition of the disease takes place usually in the members of one generation. Rutimeyer observed no less than four collateral branches of the same stock simultaneously affected. There are several other nervous diseases which attack brothers and sisters; for instance, pseudo-hypertrophic muscular paralysis, progressive muscular atrophy, Thomsen's disease, colour-blindness, and a disease of the yellow spot lately observed by Mr. Waren Tay. Most of these are also transmissible, hereditary, that is, in the strict sense, and in some of them a curious mode of propagation has been traced, viz. that the disease manifests itself in males but is perpetuated through their female descendants. Thus, a man is diseased, his sons and their descendants are exempt, his daughters are themselves exempt, but bear diseased sons. I would refer in this connection to two interesting family trees of colour-blindness and of hæmophilia, published by Dr. Wickham Legg in 'St. Bartholomew's Hospital Reports,' vol. xvii. Dr. Gee, in vol. xiii of the same 'Reports,' gives an account of a family where diabetes insipidus was propagated through the daughters. But no such definite mode of propagation has been shown to exist in Friedreich's disease. Indeed, with the exception of Carré's family, Rutimeyer's family, and the first of my

own, I do not think that ataxia has been shown to exist among the ancestry at all. Neither are females exempt from the disease; indeed, Friedreich thought they were particularly prone to it, but this may have been accidental to his observations, for amongst later cases there is a good proportion of males.

It is worth noting that these ataxic families are generally numerically large. One is tempted to think that the rapid production of children may have caused imperfect development in some of them.

Lastly, since the disease does not appear at birth, what other factors besides the family predisposition assist in its development? Friedreich held that it was connected with puberty, and doubtless this is often the case, but not invariably. I believe that the influence of acute disease is another possible factor. In two at least of my three first cases the unsteadiness followed on a feverish attack. This was described to me as rheumatic fever, but as there was no redness or swelling of the joints and no heart affection was left, it can scarcely have been rheumatic fever. There was sorethroat, feverishness and aching of the limbs, and several of the family had it at the same time. Supposing it to have been diphtheria, it would be likely enough that a complaint, which under ordinary circumstances may be followed by temporary ataxia, should, in the presence of a family predisposition, prove the starting-point of permanent spinal disease. Again, in Case 4, the symptoms were said to have become much worse after a "low fever" with sorethroat, &c., from which the mother and another sister also suffered. In Case 1, too, it was after an attack of scarlet fever that the ataxia of gait first became so marked as to be unmistakable.

[A. Woodcock family.] — Adams, drowned, æt. 40, a pilot.

Henry Adams, *probably ataxic*. Had a brother and sisters healthy (?)

Mrs. Woodcock (has had fits occasionally).

Has children—

1. Son, æt. 23, healthy.
2. Daughter, æt. 20 (Case 3), *ataxic*.
3. Daughter (Case 1), *ataxic*.
4. Son (Case 2), *ataxic*.
5. Daughter, æt. 9, healthy,[1] but *tendon reflex absent*.
6. Son, æt. 7, healthy,[1] but *tendon reflex absent or doubtful*.
7. Daughter, æt. 6, healthy; *tendon reflex not always obtainable*.
8. Son, died young of scarlet fever.

Mrs. Gravitt, *insane*.

Has children—

1. Daughter, æt. 29, married, healthy.
2. Daughter, æt. 24, healthy.
3. Daughter, æt. 21, married, healthy.
4. Son, reported to be healthy.
5. Daughter, æt. 16, healthy.
6. Daughter, æt. 13, healthy, but *tendon reflex uncertain*.
7. Son, æt. 10, healthy, but *tendon reflex uncertain*.

(Besides these, three or four either dead or stillborn.)

B. Harmer family.

No nervous disease in father or mother, or in their parents, so far as was known.

Their children are—

1. Daughter, æt. 28, married, healthy.
2. Son, æt. 24, healthy so far as is known.
3. Son, æt. 21, *ataxic*.
4. Daughter, æt. 19 (Case 4), *ataxic*.
5. Daughter, æt. 17 (Case 5), *ataxic*.
6. Son, æt. 14, reported healthy.
7. Daughter, æt. 11, *tendon reflex absent*.
8. Son, æt. 9, healthy.
9. Daughter, æt. 7, healthy, but *tendon reflex feeble*.

(The first two children that were born died of convulsions at two months and two years respectively.)

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[1] Since this paper was written both these children have shown symptoms similar to those with which the disease began in their elder relatives (July, 1885).

(For report of the discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 346.)

# FATAL HÆMOPTYSIS:

## THE STATISTICS OF THE LAST FIFTEEN YEARS OF THE CHEST HOSPITAL, VICTORIA PARK;

WITH

## REMARKS UPON PROFUSE NON-FATAL HÆMOPTYSIS.

BY

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It is an opinion now generally expressed that fatal pulmonary hæmorrhage has for its cause, with but few exceptions, some gross lesion of the pulmonary vessels, viz. either ruptured aneurysm or erosion.

It is remarkable, however, how little there is in literature beyond the record of isolated cases to support this, as I believe, quite correct conclusion. The present paper is a contribution to the subject based upon the post-mortem evidence of the past fifteen years at the City of London Chest Hospital. Dr. Douglas Powell[1] has published similar observations extending over some years at the Brompton Hospital. The only other original paper of importance I am acquainted with is that by Dr. Vald Rasmussen.[2]

[1] 'Transactions of the Pathological Society,' vol. xxii.

[2] Translated by Dr. Moore, in 'Edinb. Med. Journ.,' vol. xiv.

Although I have not introduced into the present statistics cases which I have met with in other places, the general conclusions I draw, other than statistical, are so framed as to include the experience gained from these other sources.

The statistics deal entirely with fatal cases of pulmonary hæmoptysis. After discussing these I shall consider what light is thrown by them upon cases of profuse pulmonary hæmoptysis which have not proved fatal.

By the term pulmonary hæmorrhage I wish to exclude all cases of hæmoptysis due to the rupture of a large vessel into the trachea or larger bronchi, as in the course of thoracic aneurism or new growth, and to speak only of those cases in which the hæmorrhage has its source in some portion of the lung tissue, *i.e.* of hæmoptysis in its usual limited sense.

The cases number 26, 20 males and 6 females.

| | *Age.* | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 15–20 | 20–25 | 25–30 | 30–35 | 35–40 | 40–50 | 50–60 | |
| Males . | 2 | 2 | 4 | 2 | 2 | 3 | 4 | ... 19 |
| Females | 3 | 1 | 1 | 0 | 0 | 1 | 0 | ... 6 |
| Totals . | 5 | 3 | 5 | 2 | 2 | 4 | 4 | ... 25 |

In one of the 26 the age is not given, and although the numbers are too small to justify any absolute conclusion, this table seems to show that there is no special liability at any particular age.

The earliest case was in a lad of 16, and the latest in a man of 53.

*Sex.*—It is noteworthy how much more frequent these cases seem to be in men than in women, in the proportion of 20 to 6 or $3\frac{1}{3}$ to 1.

This may be associated with the fact that the male has a greater power of resistance to phthisis than the female, or, in other words, that the disease more often becomes chronic in the male. To this point attention will be drawn again later.

*The condition of the lungs.*—Phthisis was in all cases present, and was clearly the primary cause. Cavities were always found and cavities not of recent date, but with thick fibroid walls, and ribbed or crossed by coarse trabeculæ, such as are found usually in the most chronic forms of phthisis.

Both sides were similarly affected, though not to equal degree, in 16 cases, while in 9 one side only was excavated, the opposite lung being in the condition of complementary emphysema, or, as it is often called, of compensatory hypertrophy.

These very chronic cavities seem to constitute a group of phthisis quite apart from the other and more ordinary form. In these the disease develops very slowly, often insidiously, so that there may be perhaps no history of any previous illness, and death sometimes occurs quite unexpectedly from hæmoptysis in patients who have been thought, and have appeared, in good health. Indeed, in this class of cases profuse hæmoptysis seems to be the risk most to be dreaded.

In other words, profuse hæmoptysis generally occurs in chronic phthisis, or from chronic cavities, very rarely in the subacute, and possibly never in the acute form of the disease.

*Cause of hæmorrhage.*—The source of the hæmorrhage was found in 16 cases out of 25, and proved to be aneurysm in 11, and ulcerated vessel in 6.

In the cases in which I have myself made the examination I have only twice failed to find the source, and one of these cases of failure is not included in the present number.

Too much stress must not be laid upon the term used, for ruptured aneurysms sometimes look like ulcerated vessels, especially when, by the force of the blood-stream, the whole or nearly the whole of the sac has been torn away, and I further observe in the post-mortem notes of recent years that aneurysm is a term more commonly found than ulcerated vessel, while in some of the earlier

notes the diagnosis of ulcerated vessel has the explanation added, "No saccular dilatation found," but for the reasons given the explanation is hardly adequate.

In my own cases I have only once or twice seen ulcerated vessels among many cases of aneurysm, and Rasmussen says that he has himself never met with an ulcerated vessel as distinguished from aneurysm. The difference is therefore probably in most cases rather one of terms than of reality.

*The side.*—The hæmorrhage came from the left side for certain in 11 cases, and with probability in 5 more; and from the right side for certain in 6, and with probability in 3 more. So that the left side is more often the source of the hæmorrhage than the right in the proportion of 16 to 9, or nearly 2 to 1.

When the chronic excavation before referred to is limited to one side it appears to be also most frequently on the left side, in the proportion in the present cases of 6 to 2. I know of no other statistics on this point, but this conclusion quite agrees with other observations of my own.

It does not, however, necessarily follow that the source of the hæmorrhage is found upon the side most affected, or if so in the most diseased part of it.

*The seat.*—Any chronic cavity, whatever its size, may be the source of the hæmorrhage, from a small single cavity—the only spot of disease, it may be, in the lung—not larger than a filbert, to an enormous cavity, produced by the complete excavation of the whole lung.

The cavity from which the hæmorrhage came was in the upper lobe 9 or probably 10 times, in the middle lobe twice, and in the lower 7 times, and of these last it was found 6 times in the apex of the lower lobe. In two cases the whole lung was excavated.

In the majority of cases, therefore, the hæmorrhage came from the upper lobe. The next commonest source was the apex of the lower lobe, or in the middle lobe. The lower part of the lower lobe is an unusual position.

The favourite seat, therefore, is the middle of the lung laterally, and near the periphery, whether in the lower part of the upper lobe, or the upper part of the lower. This is also the spot at which perforation most frequently occurs in pneumothorax, and it is interesting to associate these two facts together.

*Aneurysm of the pulmonary artery.*—Rasmussen, in his paper, draws a distinction between aneurysms, *i.e.* definite sacs or pouches, and ectasias or dilatations. I do not think it desirable or necessary to make this distinction, for the two classes are differentiated by no fixed characteristics, and there is every transitional stage between the one and the other, so that it is simpler to regard them as different degrees of the same affection, and to speak of them all as aneurysms.

These aneurysms spring always from a branch of the pulmonary artery, sometimes from one of the main divisions, but more often from a medium-sized branch, though frequently at only a very short distance from the origin of the smaller branch from the main trunk, so that a bristle may pass quite easily at once into the main vessel. These facts are of importance, I think, as bearing upon the origin of these aneurysms. They develop, as a rule, in the longitudinal axis of the vessel, away from the main trunk and into the lung-cavity.

They are usually found upon a trabecula which forms a more or less prominent ridge in the walls of the cavity; sometimes, though rarely, upon a trabecula which crosses the cavity, though Rasmussen says that he has never seen this latter position, and it is certainly rare. Occasionally there is no indication of the trabecula, but the aneurysm projects at once from what appears to be the smooth wall of the cavity.

The trabeculæ are the remains of the indurated vessels and bronchi of the lung, and are largest towards the root of the lung. Hence the aneurysms also are found in the part of the cavity nearest to the root. Cavities, therefore, in which aneurysms are being searched for, may be

opened through the pleura, *i.e.* from the periphery, without risk of destroying the aneurysm.

Their *shape* is, as a rule, more or less globular, extending from one side of the vessel. Those of large size have frequently secondary pouches or sacculations upon them, and sometimes look almost like a mulberry. At other times they form more irregularly oval swellings, also with secondary sacculations. This is the condition to which Rasmussen gives the name of ectasias. True fusiform aneurysms are, I think, from the nature of things almost impossible.

They are usually small, sometimes not larger than a pea, only very rarely larger than a Morella cherry, *i.e.* $\frac{1}{2}''$ to $\frac{3}{4}''$ in diameter. Aneurysms as big as a walnut are very unusual. I have exhibited two of this size; one of them, oval in shape, measured one inch and three quarters long and one inch wide. I have never met with a description of a larger aneurysm than this. There is no relation between the size of the cavity in the lung and the occurrence or size of an aneurysm. The cavity may be so small as to be completely filled by the aneurysm, and that too when other much larger cavities are present. In two of the cases recorded here, the cavity was formed by excavation of the whole lung, while the aneurysm was of small size.

Aneurysms are in most cases single. This is remarkable, but instances of multiple aneurysm are recorded by many observers. One of the cases in these tables had several.

With regard to *clotting*, Rasmussen states that pulmonary aneurysms are never found to contain laminated clot. Further observation proves the statement to be incorrect. It is, however, true that they frequently do not. The larger aneurysms generally do according to my experience, and many of the smaller ones may. Dr. Percy Kidd's case is a notable instance of this, for nearly every one of the numerous aneurysms there found was occupied by laminated clot, which in some nearly filled the cavity completely.

Many of the solid lumps found projecting from the

walls of chronic cavities are, I believe, aneurysms, which have become obliterated or cured by clotting, in the same way that aneurysms cure elsewhere, but clotting does not necessarily prevent rupture in pulmonary any more than in other aneurysms.

Clotting often occurs not only in the sac, but also after rupture outside it in the cavity, and this clot, too, is sometimes laminated. The size of the aneurysm thus becomes sometimes deceptive, and it appears much larger than it really is.

This clotting may explain the cases of remittent hæmoptysis, but though essentially a conservative process, whether within or without the sac, it does not necessarily prevent a fatal result.

The seat of *rupture* is generally at the periphery, *i.e.* at a point distant from the vessel from which it springs, but occasionally it is found at the base of the sac, as it were between the sac and the vessel.

The aperture is sometimes small, and may then be easily closed by clotting; at other times it is irregular and large, and in some cases the whole sac or the greater part of it is torn bodily off, and the rent into the vessel is represented by the origin of the sac. Some of these cases would look very like simple erosion of the vessel. In some of my own cases, I have only been able, on careful examination, to satisfy myself that I had an aneurysm, and not an erosion to deal with.

As in aneurysms elsewhere, the final rupture rarely comes without warning. In most cases there is, or has been within recent periods, some premonitory hæmorrhage. This clinical fact, associated with the known pathology, shows the importance of early and strict treatment of even slight hæmorrhage in chronic phthisis.

Rasmussen draws a distinction as to the access and the amount of hæmorrhage between the two classes which he makes of aneurysm and ectasia, but I do not think so sharp a distinction is warranted either by clinical experience or by pathological observation.

The *pathogenesis* of pulmonary aneurysm is, I think, simple. There is no case recorded, so far as I am aware, of an aneurysm of the pulmonary artery developing in an otherwise healthy lung. It is conceivable that atheroma or syphilitic disease, or some other primary affection of the pulmonary artery might lead to aneurysms, but such conditions are, to say the least, very rare. Hence we are justified in connecting the development of the aneurysm with a pre-existing cavity in the lung as already shown.

The formation of the aneurysm may be attributed:

I. To changes set up in the walls of the vessel, of an inflammatory or degenerative nature, by direct extension from the walls of the cavity.

In nearly all acute inflammatory or rapid cases clotting occurs in the vessels of the neighbourhood, and they are early obliterated. When the process becomes circumscribed, and a chronic cavity is formed, it frequently happens that a vessel of larger size remains unobliterated, and that its coats become subsequently involved in the same fibroid change which had occurred in the walls of the cavity. If the vessel remain patent the blood pressure within slowly distends the fibroid part to form a pouch.

The growth of the aneurysm is still further aided by:

II. The want of support upon the side towards the cavity, so that this side of the vessel is not only the weakest in itself, but also the direction of least external resistance.

It is, moreover, not unusual to find the vessel from which the aneurysm springs constricted or even perhaps completely obliterated peripherally, a condition which tends still further to promote the formation of aneurysm, by making the same spot also the point of maximum pressure from within.

These facts explain the association of aneurysm with chronic cavities only.

*Ulceration of vessels.*—Where a cavity is of more recent formation and has not passed into the chronic stage, or

where in a chronic cavity ulceration has set in, the change in the walls of the vessel are of a more acute character. They too may become involved in the ulceration, and after considerable thinning may rupture. But in most cases this is prevented by clotting within, and the vessel is quickly obliterated. This is so common in phthisis that occlusion of vessels has been regarded as one of the most characteristic features of the disease. If this were not so, hæmorrhage ought to be even more frequent than it is, and fatal hæmoptysis one of the commonest causes of death. The contrary is, however, the case. The percentage of deaths from hæmoptysis in phthisis is small, probably not more than 1 or 2 per cent. of all the fatal cases. My own statistics give about 1·5 per year, or about 2 to 2·5 per cent. of deaths from all causes in phthisis.

Although the pulmonary artery is the vessel usually affected in both aneurysm and ulceration, still the pulmonary vein may be attacked. I have met with one case of this kind in which the death was due to ulceration of a branch of the pulmonary vein, but it is the only case I know of.

In conclusion, aneurysm being a thing of slow growth, can arise only in chronic cases. In early phthisis hæmorrhage is probably due to ulceration or erosion of vessels, but this is rarely copious and very seldom fatal, for phthisis as a rule seals the vessels as it invades them.

When hæmoptysis leads to death, it does so in one of two ways, either by frequent recurrence of hæmorrhage and gradually increasing exhaustion, as occurs in similar recurrent hæmorrhage from other parts, or suddenly in a few moments. To this latter group the name of suffocative hæmoptysis is given. Occasionally, however, the sudden result is due to cardiac syncope, though as a fact this more frequently occurs as the final cause of death in the cases belonging to the first group, *i.e.* where death is the result of exhaustion.

The question now arises: "Are the causes of profuse pulmonary hæmoptysis the same in the non-fatal as in the fatal cases?"

To this question an affirmative answer must, I think, be given without hesitation, and for the following reasons:

1. Cases recover which can in no way be distinguished clinically from those which die.

2. In cases which have lingered and finally died from exhaustion (remittent or intermittent hæmoptysis) the two lesions described, viz. aneurysm or ulceration, have been found.

3. We have sufficient pathological evidence that pulmonary aneurysms may, like aneurysms elsewhere, spontaneously cure, and though it may be urged as an objection that if this were true, cured aneurysms should be frequently found in cases of phthisis, I do not think the objection of much weight, for they are often not looked for, and even when looked for carefully are not easy to find, while it is possible that many of the firm fibrous masses frequently found and described in chronic cavities, and of which no very satisfactory pathological explanation is usually forthcoming, may be really aneurysms obliterated and cured. Several such cases have been recently described.

4. The evidence is still stronger in favour of the cure of ulcerated vessels. In acute phthisis the vessels are at once considerably involved, and the wonder is not that profuse hæmoptysis occasionally occurs, but that it is not much more frequent. It is prevented, we know, by the thrombosis and obliteration of vessels which advance *pari passu* with the disease, and the same process continues also in chronic phthisis, in which, as is abundantly proved, ulceration of vessels is very rarely the cause of fatal hæmoptysis.

Nor are the cases of phthisis ab hæmoptoë, *i. e.* those cases in which profuse hæmoptysis is the first or earliest recognisable symptom of the disease, necessarily opposed

to the present views of hæmoptysis, for it is well known that disease may exist in the lung which no physical examination can detect, and that it is often of a latent, insidious or chronic kind, and that aneurysms have been found in such cases.

The doctrine of phthisis ab hæmoptoë can hardly I think be accepted except in the sense of post and not propter hæmoptoem; and though it is true that after hæmoptysis destructive lesions may advance with great rapidity in the lung; it is, however, equally true that the hæmorrhage sometimes not only does no harm but may even seem to do good.

It would carry me beyond the scope of this paper were I to bring forward evidence from the clinical records of cases to establish each of these assertions, but upon the answer given to these questions will depend largely the methods of treatment we pursue, and as rational therapeutics must rest upon the basis of correct pathology their importance is self-evident.

| Date. | Name and reference. | Age. | Sex. | Cause of hæmoptysis. | | | | | Condition of lungs. | Side most affected. | Cavity from which hæmorrhage came. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Ulceration. | Aneurysm. | | | Rupture. | | | Side of chest. | Size. | Position. |
| | | | | | | Number. | Size. | | | | | | |
| 1870 | John Parker (Dr. Ward, P. M. R., ii, 205) | 28 | M. | — | Aneurysm | 1 | Small walnut, artery = 1/16″ diam. | Irregular laminated clot | Chronic phthisis in both; many irregular cavities, with fibroid walls | Right | Left | Pullet's egg | Middle third of lower lobe |
| 1871 | Henry Barnes (Dr. Birkett, P. M. R., iii, 11) | 41 | M. | ? Ulceration; "no saccular dilatation found" | — | — | — | Irregular | Chronic phthisis of right upper lobe, with complementary emphysema of rest of right and whole of left | Right | Right | Large excav. | R. upper lobe, upper and inner part |
| 1872 | James Kelly (Dr. Andrew, P. M. R., iii, 18) | 36 | M. | ? Ulceration; ditto | — | — | — | Irregular | Chronic excav. of upper part of both lungs, with great fibroid induration | Equal | Left | Large excav. | Upper lobe |
| „ | John Baxter (Dr. Bennett, P. M. R., iii, 43) | 52 | M. | ? Ulceration | — | — | — | Irregular | Phthisis of both sides, with soft-walled cavities and caseous masses | — | Right | — | — |
| „ | Arthur Wilson (Dr. Andrew, P. M. R., iii, 60) | 17 | M. | ? Ulceration | — | — | — | Irregular | Large chronic cavity in right upper lobe; recent caseous change in left | Right | Right | — | Upper lobe |
| 1874 | Edwin Cordery (Dr. Peacock, P. M. R., iii, 86) | 30 | M. | No source found | | — | — | — | Large cavity in left apex, walls not very fibrous; rest of left and also right contained caseous masses and a few small cavities | Left | ? Left | — | Apex |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| „ | Sarah Smith (Dr. Andrew, P. M. R., iii, 88) | 19 | F. | No source found | — | — | — | Chronic excavation in left upper lobe, with recent caseous change in rest as well as in right lung | Left | ? Left | — | Upper lobe |
| 1876 | Thomas Smith (Dr. Birkett, P. M. R., iii, 139) | 53 | M. | No source found | — | — | — | Chronic phthisis; both lungs in a condition of slaty induration, with cavities with fibroid walls | — | ? Right | — | Probably from middle lobe |
| „ | Geo. Carpenter (Dr. Birkett, P. M. R., iii, 151) | 27 | M. | No source found | — | — | — | Cavity with fibroid walls in left apex; right emphysematous | Left | Left | — | Probably apex |
| „ | Joseph Larman (Dr. Birkett, P. M. R., iii, 152) | 21 | M. | — | Aneurysm | 1 | Hazel nut | — | Extensive excavation of upper two thirds of left lung; right emphysematous | Left | Left | Walnut | Middle of apex of lower lobe, just below pleural septum |
| „ | Emma Harris (Dr. Andrew, P. M. R., iii, 167) | 17 | F. | — | Aneurysm | Several | Small | Small | Left lung converted into one large cavity, with several aneurysms upon trabeculæ; right lung also contained several cavities with fibroid walls | Left | Left | Whole lung | — |
| „ | Susan Bissett (Dr. Peacock, P. M. R., iii, 172) | 44 | F. | Ulceration; aperture partly occluded by clot | — | — | — | Irregular | Phthisis, with recent mischief in both lungs, most advanced in left; in apex of left a large cavity; empyema on right side | Left | Right | Pigeon's egg | Middle lobe |

| Date. | Name and reference. | Age. | Sex. | Cause of hæmoptysis. | | | | | Condition of lungs. | Side most affected. | Cavity from which hæmorrhage came. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Ulceration. | Aneurysm. | | | Rupture. | | | Side of chest. | Size. | Position. |
| | | | | | | Number. | Size. | | | | | | |
| 1878 | Joseph Wood (P. M. R., iv, 73) | ? | M. | Ulceration | — | — | — | Irregular | Chronic excavation in left apex; right lung emphysematous | Left | Left | — | Apex |
| 1879 | Thomas Waldie (P.M.R., iv, 153) | 50 | M. | — | Aneurysm | 1 | — | Irregular | Chronic excavation of both apices, most in left | Left | Left | — | Up. lobe, outer part |
| 1880 | John Harrington (P.M.R., iv, 190) | 38 | M. | — | Aneurysm | 1 | ½ inch in diam. | Half torn across | Complete excavation of left lung, with emphysema of right | Left | Left | Whole lung | — |
| 1881 | James Doyle (P. M. R., v, 1) | 27 | M. | — | Aneurysm | 1 | ½ inch in diam. | Irregular | Excavation of whole upper lobe of left, with chronic induration and cavities of lower lobe; complementary emphysema of right | Left | Left | Cherry | Apex of lower lobe |
| „ | Agnes Pyman (P. M. R., v, 27) | 20 | F. | Source not found | | — | — | — | Chronic excavation of right upper lobe; complementary emphysema of left lung | Right | Probably left | — | — |
| „ | Mary Ford (P. M. R., v, 48) | 19 | F. | Source not found | | — | — | — | Chronic, induration, with fibroid walled cavities in both | Left | Probably left | — | — |
| 1882 | Sarah Payne (P. M. R., v, 72) | 29 | F. | — | Aneurysm | 1 | ½ inch in diam. | — | Chronic cavities in both lungs | Equal | Right | Small | Apex of lower lobe |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1883 | John Ashley (P. M. R.,v, 103) | 55 | M. | Source not found | | — | — | — | Chronic cavities, with fibroid walls in both apices; largest left | Left | Left | Large | Apex |
| „ | Alfred Vickery (P. M. R.,v, 105) | 26 | M. | — | Aneurysm | 1 | Small cherry | Irregular near base | Large chronic cavity in left apex; smaller ones in upper part of lower lobe; chronic induration of right lung, but no cavities | Left | Left | Small | Apex of lower lobe |
| „ | George Thrussel (P. M. R., v) | 40 | M. | Source not found | | — | — | — | Chronic phthisis of both | Equal | Probably right | — | — |
| „ | Bartholomew Haye (P. M. R., v) | 34 | M. | Source not found | | — | — | — | Chronic excavation of left upper lobe; chronic cavities of smaller size in right | Left | Probably right | — | — |
| „ | Geo. Macarthy (P. M. R., v) | 40 | M. | — | Aneurysm | 1 | Oval, 1¼″ by 1″ | Irregular 1″ long in axis of vessel | Chronic excavation of right upper lobe; chronic cavity in apex of lower lobe; complementary emphysema of left lung | Left | Left | Tangerine orange | Apex of lower lobe |
| „ | Thomas Whittingham (P. M. R., v) | 16 | M. | — | Aneurysm | 1 | Small cherry | Small and irregular | Numerous small chronic cavities in both lungs, largest in apex of left | Left | Left | — | Apex |

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 350.)

# ACUTE PERITONITIS TREATED BY ABDOMINAL SECTION.

BY

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AMONG the great changes that have of late years taken place in the practice of surgery there are probably none more remarkable or more revolutionary than those that concern the serous membranes. Within quite recent times serous cavities were held by surgeons in a kind of respectful dread—they were sacred enclosures, into which the knife of the boldest seldom ventured; they held an immunity from all forms of active interference and were accounted exempt from the rudimentary principles of surgical treatment. With regard to the larger cavities—those of the chest and abdomen—it is interesting to note how abruptly operative procedures ceased at the confines of the pleura and peritoneum. Disease was pursued by the knife up to the very walls of these cavities, but when it had found a place within the enclosure itself it had found a sanctuary into which the surgeon did not trespass.

The most remarkable advances of modern surgery are

based upon a disregard of the supposed peculiarities of the serous membranes and—following after this—upon the application to serous cavities, and the viscera they contain, of the common general principles of surgical practice. Ovariotomy is founded upon the same principle that advises the removal of a tumour of the surface; extirpation of a disorganised kidney upon the same principle that urges the excision of a disorganised eyeball; and excision of an epithelioma of the colon, or pylorus, upon the same principle that sanctions the removal of an epithelioma of the lip. It cannot, indeed, be said that the surgery of the chest and abdomen, as it now appears, involves any new element in the treatment of disease. The most striking of the operations that have of late years been directed against the serous cavities and their viscera, consist at the best merely of the application to those parts of common principles of treatment that have long been applied to other portions of the body.

It is to the treatment of inflammatory affections of serous membranes that I would venture to especially draw attention.

In the management of inflammatory conditions of superficial parts or—it may even be said—of parts beyond the confines of the serous membranes, it is the common and most ancient practice (when milder means have failed) to freely incise the involved part, for the purpose of relieving congestion and tension, and more especially for the purpose of giving a free escape to all inflammatory products. It is only within comparatively recent times, and only in a progressive and cautious manner, that surgeons have applied this common principle to the treatment of inflammations of serous cavities. They began in the first place with small serous cavities—with the joints. The disordered joint was cut into, the inflammatory matters that it contained were evacuated, it was washed out and drained, and the result was satisfactory. One has not to go far back in the history of surgery to find a time when the treatment of a synovitis of the knee by

incision and drainage, and a washing out of the joint, would be considered a violent and reprehensible measure. Next in turn came a much larger serous membrane—the pleura. The inflammatory products of pleuritis were at first cautiously removed by tapping; gradually the common principle of treatment was extended, and the pleural cavity was cut into; it was washed out and drained. The results were good, although the means employed would have struck terror to the heart of the ancient surgeon.

Quite recently, even the pericardium has been attacked and suppurative pericarditis has been treated upon the same principles that direct the treatment of suppurative affections elsewhere.

Finally, this common therapeutic measure has been applied to the greatest of the serous membranes—the peritoneum. Cases of chronic localised peritonitis, and especially of pelvic peritonitis, have been treated with considerable success by means of abdominal section, with subsequent irrigation, and drainage of the suppurating cavity.

Considering the very high death-rate of acute diffused peritonitis, and, indeed, the almost invariable fatality of that disease, it appeared to me that the common surgical measure above alluded to might well be applied to this affection.

Its success in chronic localised peritonitis appeared to encourage its application to the more violent and rapid form of inflammation.

Apart from some such active treatment it must be confessed that the treatment of acute peritonitis, and particularly of that form depending upon perforation, remains in about the same condition at the present time that it held a hundred years ago. Thus Heister, writing in 1739 on perforation of the bowel, could only advise that the patient be kept quiet, that he be urged to eat abstemiously, and to lie upon his belly. "The rest," says this ingenious author, "is to be left to Divine Providence and the strength of the constitution."

I might now give details of a case of acute diffused peritonitis that was treated with success by abdominal section. It is the only case upon which I have performed the operation, and I am not aware that this form of peritonitis has hitherto been deliberately subjected to this particular proceeding.

A single woman, æt. 20, was admitted into the London Hospital, under the care of my colleague, Dr. Herman, on January 21st, 1884. For twelve months she had led an immoral life, and had acquired gonorrhœa. On admission she was found to be suffering from chronic pelvic peritonitis, which appears to have commenced two months previously and to have been due to the extension of the gonorrhœal inflammation to the uterus and Fallopian tubes. She was weak, wasted, and anæmic, and had a purulent vaginal discharge. She had much pain and tenderness in the left iliac region, and in that position a slight swelling could be detected, which appeared to extend down into the anterior *cul-de-sac*. There was no general abdominal tenderness; her bowels acted regularly without medicine; she took her food fairly, and was not sick. Her temperature ranged between 99° and 101° or 102°. She remained in this condition, getting neither better nor worse, for about a month, and on February 25th she suddenly developed the evidences of acute peritonitis. When I saw her on the following day, at Dr. Herman's request, I found her greatly prostrated. Her abdomen, which had hitherto been flaccid, was now very tense, evenly distended and tympanitic, and exceedingly tender. Her bowels had ceased to act. She was troubled with almost constant vomiting, and could retain nothing on her stomach. The ejected matters had an unpleasant intestinal odour. Her pulse was very small, weak, and frequent, and her tongue dry. She had had morphia. Her condition being very critical, I arranged at once to open the abdomen, which I did on the afternoon of February 26th. I made an incision in the linea alba below the umbilicus large enough to admit my hand. On open-

ing the serous cavity a quantity of semi-opaque fluid gushed out, which was mixed with flakes of lymph and pus. It was very offensive and had a decided feculent odour. It ran out on to the floor, and the amount therefore could not be measured. I then gently introduced a sponge, and cleared out a sufficient amount of the remaining fluid to enable me to see the condition of the parts. Examination showed localised pelvic peritonitis on the left side; this had led to a large abscess, the walls of which were formed partly by the left pelvic peritoneum and partly by many coils of small intestine which were matted together in the utmost confusion. The slight swelling previously felt in the left iliac region had been apparently caused by this mass of intestines, while that detected in the anterior *cul-de-sac* had been due to the purulent collection. The abscess that had been so long hemmed in by a barrier of adherent intestines had burst, at last, into the general peritoneal cavity, and had so set up acute and general peritonitis. I enlarged, with my finger, the opening into the abscess, and sponged out a large quantity of thin stinking pus, which welled up on pressure from the depths of the pelvis. Such adhesions between the matted bowels as could be broken down I tore through. The majority were too tough. The general surface of the peritoneum exhibited the ordinary appearances of acute peritonitis. The intestines were lightly glued together where they were in contact with one another. I now proceeded to thoroughly wash out the whole peritoneal cavity. I poured in many quarts of warm water mixed with a little carbolic solution. The pelvis was well sponged out. I continued the cleansing process until the water returned quite clear and free from smell. A large drainage-tube, six inches in length, was passed down to the bottom of the pelvis, and the abdominal wound—save the part occupied by the tube—was carefully closed. The operation was conducted under strict Listerian precautions, and the usual gauze dressings applied. The patient only vomited twice after the operation, probably from the

effects of the ether. No food of any kind was administered by the mouth for four days, during which time morphia was freely given and the strength supported by enemata of peptonized beef-tea and brandy. These enemata were administered every two or three hours and were readily absorbed. The bowels acted spontaneously on the fifth day after the operation. For four days the patient suffered from carboluria. There was a very copious purulent discharge from the wound, which for nearly a fortnight was dressed twice a day. The antiseptic dressings were discontinued on the fifth day. The carbolic acid produced severe irritation of the skin, and apparently caused the edges of the wound to slough. After the fifth day iodoform gauze was used; and the wound was well irrigated with very weak carbolic lotion twice or three times every day. The tube of the irrigator was passed deep down into the pelvis. The wound healed all but the part occupied by the drain; the discharge gradually diminished, and the patient underwent a very rapid and remarkable improvement. In time a very small drainage-tube took the place of the larger one, and the discharge diminished to about two drachms in the twenty-four hours. By April 5th the patient was allowed to walk in the garden. On May 3rd the tube was removed and the wound allowed to close. She was kept under observation until June 6th, when she was finally discharged. In August last she again came to the hospital. She had just menstruated for the first time since her illness; the process had been attended with severe pain and vomiting. A few days afterwards the thin scar over the site of the old drainage-hole gave way and some pus again escaped. The urgent trouble, however, soon subsided, and she went out with a minute tube in the wound to prevent its healing, although the amount of discharge barely soiled a piece of lint in the course of twenty-four hours.

With the effect of the operation upon the original pelvic trouble the present inquiry is not directly concerned; nor has sufficient time elapsed for that question to be de-

cided. I think that it may, however, be claimed that the treatment of the acute affection was successful, and that the abdominal section in every probability saved the patient's life.

In many instances the abdomen has been opened in cases of acute perforative peritonitis where an error in diagnosis had been made and the affection mistaken for intestinal obstruction. Duplay has collected several of such cases.[1]

In many reported instances, moreover, laparotomy, undertaken for some independent disease, has been performed, during the progress of an acute peritoneal inflammation. I am not aware, however, that abdominal section has been deliberately adopted as a means of treatment in this acute affection.

So long ago as 1848 Mr. Hancock, in a paper read before the Medical Society, threw out a proposal for this method of treating peritonitis, and presumably he included the acute as well as the chronic form. Mr. Hancock has described a case of localised chronic peritonitis, following disease in the appendix, in which he had evacuated a quantity of inflammatory matter through an abdominal incision. He concludes his comments with this remark, "I trust the time will come when this plan will be successfully employed in other cases of peritonitis."

Of the instances above alluded to, of laparotomy performed during acute peritoneal inflammation, owing to an error in diagnosis, death appears—with one exception—to have been the result in all. They were principally cases of acute perforative peritonitis, and as soon as the nature of the case was rendered evident by the laparotomy, all further treatment was abandoned, and the case left, as Heister would say, "to Divine Providence and the strength of the constitution." The exception alluded to was afforded by a most interesting case recorded by Dr. Buchanan, of Glasgow.[2] The symptoms were believed to

[1] 'Archives gén. de Méd.,' 1879, p. 207.

[2] 'Lancet,' vol. i, 1871, p. 776.

be due to intestinal obstruction. The abdomen was opened; no cause of obstruction was found, but, on the other hand, an extensive acute peritonitis. The abdominal cavity was sponged out, and the patient recovered. The cause of the serous inflammation was not evident, and the precise nature of the case is a little obscure.

With regard to the instances where laparotomy has been performed during the height of acute peritonitis, for some intercurrent disease, it must be confessed also that death has followed in the majority of the cases. In many of these examples, however, operative interference was undertaken when the patient was *in extremis*, and no especial treatment was directed against the peritoneal inflammation.

There are at the same time some notable instances of recovery under this circumstance. As examples I might cite two cases, one by M. Terrier,[1] the other by Professor Juillard.[2] In the former a laparotomy was performed to relieve a strangulation of the gut, by a band; in the latter an ovariotomy was undertaken in a case where symptoms of acute intestinal obstruction had suddenly set in. In both there was acute general peritonitis, in both strict Listerian precautions were adopted, in both a perfect recovery followed.

Another most successful case is reported by Mr. Thomas Keith, of Edinburgh.[3] Here an ovariotomy was performed when the cyst was gangrenous, and when distinct evidences of acute peritonitis existed. This bold operation of Mr. Keith's is, so far as I know, the first of its kind in this country.

I have selected these three cases because they present another common feature of considerable interest. The three patients had suffered from previous attacks of peritonitis of a localised or chronic form. I believe that this latter circumstance contributed in no small degree to the final

[1] 'Bull. et Mém. de la Soc. de Chir. de Paris,' 1879, p. 564.

[2] Ibid., p. 627.

[3] 'Lancet,' vol. ii, 1865, p. 86.

success of the operations. The peritoneum would appear to become in time almost acclimatised to inflammatory attacks, and there is certain evidence to show that a peritoneum that has once been inflamed will bear operative interference with greater indifference than will a membrane upon which no such morbid process has encroached.

It is to be noted that, in my own case, the patient had had somewhat extensive chronic peritonitis before the acute attack, for which the abdominal cavity was opened, set in.

In conclusion, I would venture to suggest the use of abdominal section in the treatment of acute general peritonitis; the incision to be followed by irrigation of the serous cavity, and subsequent drainage.

The cases for which this measure would appear to be best adapted are those of acute peritoneal inflammation, depending upon sudden perforation by ulcer, or by gunshot or other wound, cases due to injuries other than those producing perforation, and examples of acute peritonitis due to the bursting of an abscess into the serous cavity.

In all such instances the incision, if it be made at all, should be made without delay, and, indeed, as soon as possible after the diagnosis has been distinctly established. It would be difficult to name a condition in disease where temporising would be more utterly futile, or more reckless, or more entirely purposeless. The grave character of the affection would render almost any measure justifiable. Indeed, the lover of the curious will have to search far into the records of medicine before he will find an instance of recovery from acute perforative peritonitis; and in a case so urgent and so desperate it can hardly be a violent or unreasonable act to apply to the relief of peritoneal inflammation the same measures that have been so successful in the cure of inflammations of other parts.

Dr. Parkes, of Chicago, has shown by a series of valuable experiments how much can be done to save life in

cases of perforation of the intestine by gunshot wounds. This surgeon shot a number of dogs through the abdomen. He allowed an interval to elapse during which extravasation of intestinal contents could take place. He then opened the abdomen, closed the perforations in the bowel by suture or resection, cleaned the peritoneal cavity and united the parietal incision. By this means he saved the lives of many dogs who would otherwise have died without doubt of acute perforative peritonitis.[1]

Professor Kocher, of Berne, has performed laparotomy in a pistol-shot wound of the stomach with success. The operation was performed three hours after the injury, and the wound in the stomach was closed by sutures. Dr. William Bull has also published[2] a most successful case of laparotomy for gunshot wound of the intestine. The holes in the intestine were closed by sutures and the peritoneal cavity washed out. The operation was performed seventeen hours after the accident, and there were already evidences of extensive peritoneal inflammation.

There are, of course, cases of acute inflammation of the peritoneum, to which this mode of treatment, or any other measure of like purport, would not be applicable. Among such may be named peritonitis in connection with carcinoma, or tuberculosis of the serous membrane, the peritonitis that is the outcome of general septicæmia, and that attended with extensive rupture of certain of the viscera.

[1] 'Gunshot Wounds of the Small Intestine,' Chicago, 1884.

[2] 'New York Medical Journal,' Feb. 14th, 1885.

(For report of the discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 362.)

# A CASE

OF

# ABDOMINAL SECTION

FOR

# ACUTE CIRCUMSCRIBED PERITONITIS.

## RECOVERY.

BY

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Received March 3rd—Read March 10th, 1885.

J. R—, a medical student, æt. 19, was admitted into St. Bartholomew's Hospital, on December 18th, 1884, under the care of Dr. Andrew. He stated that, though subject to occasional constipation, he had been in good health till December 8th, when, having taken some aperient medicine, he was attacked with severe griping pain, and with sickness which had continued till his admission. The bowels acted several times after the medicine, but for the last three days nothing had been passed. When first seen he lay with his legs extended. The abdomen was tympanitic and distended, and slightly tender. A little below and to the left of the umbilicus there was an area about eight inches square, which was firm and resistant,

dull on percussion, painful on pressure, and yielding an obscure sense of deep-seated fluctuation. This area was slightly raised above the surrounding surface, and the skin was œdematous and presented a slight flush. As it was believed that the case was one of circumscribed peritonitis, it was on the following day determined to perform an exploratory operation. Just before the patient was removed to the operating theatre he was violently sick several times, and it was obvious that his condition was rapidly becoming very critical. When he was under chloroform, I made an incision about two inches long just outside the linea semi-lunaris, and opened the cavity of the peritoneum. As soon as this was done, from two and a half to three pints of extremely fœtid pus, evidently under considerable tension, streamed out. A careful digital examination was now made, but nothing beyond distended coils of small intestine could be felt. The abscess-cavity was washed out with a solution of carbolic acid lotion (1 in 60) introduced by means of a soft india-rubber catheter, and a piece of drainage-tube eight inches long and about the size of a little finger, was inserted through the wound, the upper and the lower ends of which were brought together to avoid the danger of intestinal protrusion. Carbolic dressings were applied, and kept in place with a lightly adjusted bandage. Two hours after the operation he was cold and collapsed, and his pulse was very quick and small; two hours later, however, he had rallied considerably, and his pulse was 96, regular, and fairly strong, and his temperature was normal. He had been sick several times, but the material vomited had quite changed its character, and now consisted merely of clear, bilious liquid, derived from the upper part of the small intestine.

December 20th.—He was decidedly better. Pulse 96, temperature normal, and he had passed a good night, sleeping quietly, with a few intervals of wakefulness. He was sick occasionally till 8 a.m., bringing up about 14 oz. of bilious fluid. In the night, at 10 p.m. and at

2 a.m. a third of a grain of morphia had been injected under the skin. Discharge was profuse, and the dressings were found to be soaked through. During the day the bowels acted three times, the motions being light coloured and fluid. He was fed with small nutritive enemata, and only allowed to take, by the mouth, a teaspoonful of iced water occasionally. He complained of intense thirst during the day. An enema of starch and opium was given to check the action of the bowels. At 6 p.m. the area of dulness could be felt to extend across the middle line towards the right iliac fossa, and in this situation he complained of tenderness on pressure. The nurse was directed to keep him as far as possible on his left side.

21st.—Night rather restless. Morning temperature was 99·6°. There had been no more sickness. The abdomen was less tympanitic; the area of dulness was diminished; the bowels had acted four times within the last twenty-four hours, the motions consisting of light yellow liquid, mixed with small, solid, well-formed fæces. He was taking milk and beef tea in small quantities.

22nd.—Was improving. Temperature last night was 99·6°, this morning normal. Pulse 96. As the drainage-tube was found to have slipped out it was discontinued. Still a large quantity of purulent discharge. A piece of distended intestine protruded slightly at the bottom of the wound.

23rd.—Steadily improving. Wound looked well. Temperature ranging from 100° in the morning to 101·8° in the evening. Pulse 108, small. Was still fed with enemata. He had taken about a pint of warm milk and water, and two small cups of tea, and a little bread and milk.

26th.—Decidedly better. Abdominal distension had subsided. Discharge copious, but healthy. Free from fœtor. Bowels were acting twice a day, and the motions, though in great part fluid, contained well-formed fæces. Still on liquid diet. Was occasionally taking ten minims of tincture of opium to quiet the bowels.

January 4th—Doing well. Had minced chop for dinner. For the last four days the cavity had been washed out with a solution of 1 in 1000 of tincture of iodine introduced by means of a soft india-rubber catheter. This passed easily for eight inches or more in several directions among the coils of the small intestine.

12th.—There had been a threatening of a protrusion of the intestine through the wound, but this had been prevented by the application of a compress of cotton wool over a piece of oiled lint and a lightly applied bandage. He now spent the greater part of the day on a couch.

20th.—Doing well, but his bowels now seldom acted without an enema. The protrusion of the intestine was less marked.

February 20th.—Since the last note there had been little requiring detailed description. He had gradually improved and gained flesh and strength. His temperature had been as a rule normal, and his pulse between 80 and 90. But both on Feb. 8th and on Feb. 19th he had attacks of sickness, lasting for some hours; his temperature rose to 101°, and his pulse to 96, while at the same time there was marked increase in the amount of discharge. During the past week he had improved quickly in strength and colour. The bowels now never acted without injection. The wound was slowly closing by granulation, and was only about half an inch in length.

March 7th.—Wound now closed. Bowels still never acted without an injection. He seemed quite well.

*Remarks.*—Neither Dr. Andrew nor myself could form any definite opinion as to the origin of this attack; but it seemed not unlikely that inoculation of the peritoneum had occurred from the escape of pus from an abscess formed in the mesentery round old suppurated glands.

Although the case was one of circumscribed peritonitis, that it yet involved fully a third of the whole abdominal cavity was shown by the fact that the swelling and dulness not only occupied the greater part of the left lumbar and iliac regions, but was found, as distension subsided,

to extend for several inches across the middle line in the direction of the right iliac fossa. The extent of the mischief was also disclosed by the direction and distance from which pus was washed out when the catheter was introduced. It was therefore an instance of much more extensive disease than would generally pass by the name of circumscribed, and approached the characters of an example of general peritonitis. It thus has at least some bearing on the treatment of the latter affection.

At the time the operation was performed the patient was so constantly sick, and so rapidly passing into a condition of collapse, that it was the opinion of all who saw him that unless relieved he would live but a very few hours. The operation calls for no especial comment. It consisted merely in the opening and washing out of a large intra-peritoneal abscess. It may, however, be remarked that it seemed better to rest content with the evacuation of the matter which had collected than to enter into any investigation into the origin and nature of the case. To have taken the latter course would not only have involved the danger of disturbing the adhesions which were acting as a barrier to circumscribe the mischief, but also the risk of rupturing the softened intestinal coils and producing fæcal extravasation. Besides, any investigation of this kind could scarcely have led to any useful result, while it would certainly have necessitated a considerable enlargement of the wound, and increased the possibility of an escape of some portion of distended intestine.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 362.)

# ANEURISM OF ABDOMINAL AORTA.

## DISTAL COMPRESSION—CURE OF THE ANEURISM—DEATH FROM GANGRENE OF THE JEJUNUM ON ELEVENTH DAY—NECROPSY—REMARKS.

BY

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AND

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(Communicated by Mr. R. W. PARKER.)

Received January 2nd—Read April 14th, 1885.

E. B—, æt. 32, a shoemaker by trade, was admitted into the Infirmary in October, 1883. He had been in the army nine years, and had syphilis five years ago. No history of strain or injury.

Two weeks before admittance he was suddenly seized with severe pains in his back and epigastric region; these became much worse during the night, and at the same time he noticed a pulsating swelling in the abdomen, which had not been previously observed. He continued in the same condition up to the time of admission.

On admission the patient's general appearance was healthy; there was slight hypertrophy of the heart and a

trace of albumen in the urine. He lay chiefly on his right side; there was no marked tension or tenderness of the abdomen; strong pulsation could be seen and felt between the costal cartilages of the eighth and ninth ribs in the middle line. A large tumour could be felt deep down in the epigastric region, shading off into the right and left hypochondriac and umbilical regions; moveable by the hand but not with respiration. Pulsation was distinctly expansible, and movement was greatest on the right side. There was a slight systolic murmur over the swelling. Compression of the aorta below the tumour caused pain and uneasiness in the region of the tumour, but the size and pulsation were lessened. The dimensions of the tumour were about six to seven inches from side to side, five to six inches from above downwards, and reached apparently from the vertebral column to the anterior abdominal wall. No diminution or inequality of pulsation in the femoral arteries. Bowels confined; no vomiting.

As the usual remedies, including rest, low diet, narcotics, &c., gave no relief, and the patient was evidently getting worse, he was extremely anxious that some operation should be performed.

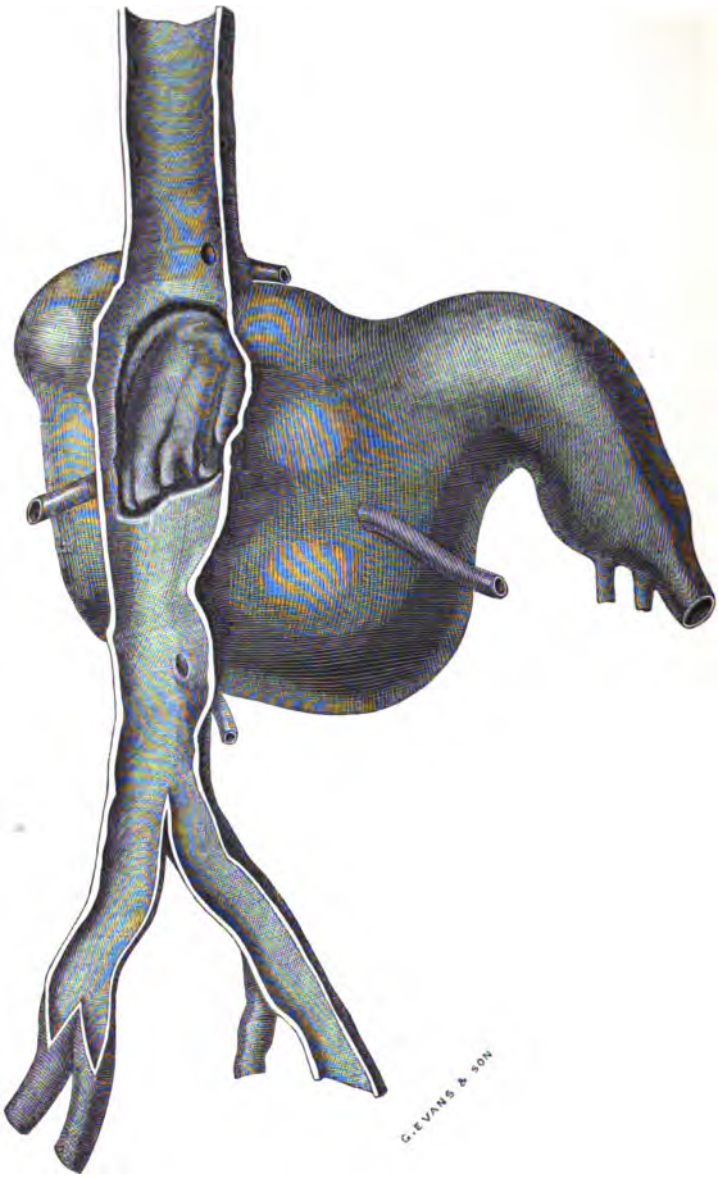
On October 31st the patient was placed under the influence of chloroform, and compression of the abdominal aorta just above and to the left of the umbilicus was commenced. Carte's tourniquet was used, and the usual precautions taken in the application and continuation of the pressure. Chloroform and ether were used alternately, and compression was applied for four and three-quarter hours. During the latter half of this period the pulse became very rapid, feeble, and irregular, and the breathing embarrassed. A small quantity of urine was drawn off by catheter, and showed a marked increase of albumen. After the completion of the operation an ice-bag was applied to the abdomen, and the patient passed a good night. On the following morning all the physical signs of the aneurism were less marked. The ice-bag was discontinued on the second day. There was slight vomiting on

the first and third day after the operation (which apparently was due to the chloroform and morphia), but otherwise the patient expressed himself as much better, and the tumour was smaller, harder, and pulsation less marked.

He continued to do well for several days, but on Nov. 8th persistent vomiting, chiefly of dark grumous material, set in. Pulse 192, and feeble; thirst was intense, there was congestion of the face, and some dulness over the right lung was detected. From this time the patient gradually became worse, and died November 11th, or the twelfth day after the operation.

*Post-mortem examination* (forty-eight hours after death).—Body fairly well nourished. Head normal. The heart was found hypertrophied, and the cavities dilated. The aorta was free from atheroma. Lungs emphysematous. Serous effusion in right pleural cavity (1½ pts.). Slight consolidation of middle third of right lung; congestion of base of left lung.

*Abdomen.*—No evidence of peritonitis. Intestines normal, with the exception of about two feet of lower portion of jejunum, which was very dark in colour in its entire thickness, had a peculiar earthy smell, and contained black grumous fluid, similar to the vomit during life. Immediately below the cœliac axis was a large sacculated aneurism, which sprang from the front of the aorta; the orifice being oval (1 × 2 inches). This was filled with a spongy red clot protruding slightly into the lumen of the vessel (see woodcut, p. 195.) The vertical diameter was about four inches, the horizontal five inches, and the antero-posterior four inches. The clot covered the front and partly the sides of the aorta; there was no erosion of the vertebræ. The duodenum curved over its anterior surface from the right upper corner to the lower margin of the sac, and was closely attached. The pancreas lay loosely on the upper surface. The left renal vein was firmly adherent, and crossed over the anterior surface about the centre of the sac. The aneurism in shape resembled a retort with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the body to the left, and the beak (origin of the superior mesenteric artery) to the front and right; the left side was fixed, and the right more moveable (see woodcut, p. 194). The branches of the cœliac axis ran along the top of the sac and were adherent to it. The phrenic and right renal arteries were given off from the sac; the left renal from the aorta just behind it. The superior mesenteric seemed to be the branch chiefly involved; it was dilated and formed a secondary aneurism. The main branch was almost, and the lesser branches completely, occluded. There was a recent loose black clot in the right iliac artery, and the deep-seated abdominal veins were much distended with blood.

*Remarks.*—The only reported cases we have been able to find in which operative treatment has been attempted, are the following:—

1. A cured case is reported by Dr. Moxon and Mr. Durham in the 'Med.-Chir. Trans.,' of 1872, vol. lv, when proximal compression by Liston's tourniquet was applied for ten hours under chloroform.

2. Mr. Bryant relates a case in the same volume which was treated by distal pressure with Liston's tourniquet for twelve hours, and after an interval of twelve hours the tourniquet was reapplied for a further period of three hours. The patient died thirty-nine hours after its first application.

3. Dr. Greenhow reports a cured case of abdominal aneurism in the 'Med.-Chir. Trans.,' vol. lvi, for 1873.

4. Dr. Murray, of Newcastle, has also reported a case cured by proximal pressure upon the abdominal aorta.

From the above list it will be seen that, with one exception, surgeons have previously chosen proximal rather than distal compression in cases of abdominal aneurism. It may therefore be of interest to draw attention to the main points in the present example. Firstly, as to the symptoms during the operation, and the result thereof, and secondly as to the conclusions that may be drawn from the case.

Firstly, there was marked alteration in the circulation, shown by a great temporary increase of albumen in the urine, presumably from the higher blood-pressure in the renal arteries, great acceleration and smallness of the radial pulse with rapidity and oppression of breathing from diminution of blood in the lower extremities, and corresponding increase in the lungs. This interference did not subside when pressure was discontinued, but persisted and gave rise to partial consolidation of the lung. This raises the question whether venesection performed shortly after the operation would have been of service in restoring the equilibrium of the blood-pressure.

The next point is the obstinate vomiting and hiccough which began on the sixth day (distinct in time and character from the early vomiting due to the anæsthetic and morphia). This persisted more or less until death, and strongly resembled the vomiting of intestinal obstruction. The most probable causes of the vomiting appeared to be—

1. *Nervous* from pressure on the aortic plexus of the sympathetic.

2. Congested state of the stomach.

3. Intestinal obstruction arising either from peritonitis, laceration of the small intestine by the pad of the tourniquet, or, lastly, gangrene of the intestine from occlusion of the superior mesenteric artery.

On careful consideration of the whole case, and remembering the character of the vomit, gangrene of the intestine appeared to be the real cause. This seems to be proved by the autopsy, for gangrene of a considerable length of the gut was obviously the cause of death, and this condition was clearly due to the arterial thrombosis. This complete blocking of the superior mesenteric artery was an unfortunate but unpreventable result of its arising from the distal part of the aneurism where the clot was firmest, as other vessels arising nearer to the upper portion of the sac remained patent.

The conclusions we draw, then, from the experience of

this case, are, that the operation was justifiable and even hopeful; that it accomplished the purpose intended; and that recovery would probably have taken place had not the process of cure been (unavoidably) too thorough; and that further, although fatal consequences must almost inevitably follow from these conditions, they cannot be recognised so precisely in cases similar to ours as to prohibit the use of the means of cure we adopted.

The specimen is presented to the museum of the Royal College of Surgeons.

(For report of the discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 426.)

# ON A CASE

OF

# ANEURISM OF THE ABDOMINAL AORTA,

WHICH CAUSED

## GANGRENE OF THE RIGHT LOWER EXTREMITY, PARTLY BY EMBOLISM, AND PARTLY BY PRESSURE ON THE INFERIOR VENA CAVA.

BY

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THE following case is an instance of what is probably a very rare cause of gangrene of the lower limb; and an example of death from an aneurism full, or nearly full, of laminated clot. It is also another illustration of the fact that advanced caries of the vertebral column may exist without any obvious clinical signs of the disease.[1]

Thos. S—, æt. 38, a carver and gilder, was admitted on January 1st, 1885, with gangrene of the right lower limb. On the 27th of December, 1884, at 6 a.m., the morning being bitterly cold, he started to go to work as usual, but

[1] Since writing the above another case of advanced spinal caries without symptoms, and the result of aneurism of the arch of the aorta has passed under my notice. The patient died suddenly whilst turning in bed from the breaking of the spine at the diseased point.

on crossing the road just outside his house, he was seized with pains, numbness, and a pricking sensation in the soles of his feet and the calves of his legs, accompanied by profuse sweating. He recrossed the road and reached his own door, grasping the railings to save himself from falling. He was, however, obliged to let himself down for rest, but after a time with much difficulty he raised himself with his hands, unlatched the door, entered the house, and again lowered himself to the ground, almost entirely by the aid of his hands. For some time he remained at the foot of the staircase, but at length he was discovered and carried up to bed by the other inmates, who applied hot water bottles to his feet and legs. During all this time both legs had seemed to him to be similarly and equally affected, but in the course of the same day he regained the mobility and sensibility of the left lower limb. On the following day (December 28th), the right leg and foot began to turn black; and continuing to get worse, he was brought to the Middlesex Hospital on New Year's Day.

On admission, he told us that sixteen years ago he had syphilis, that he had never suffered from rheumatism, nor from any lung or heart disease, though for some time past he had been subject to shortness of breath; that six months ago an attack of "lumbago" kept him in bed for about a week; and that three months ago he was taken with drowsiness and throbbing pain in the right temple and right eyeball, and could not raise his right upper eyelid. He had, in fact, temporary complete ptosis.

His father died aged forty, cause unknown; his mother aged sixty-three, from cancer of the breast; and one sister died, aged fifty-five, of heart disease.

The patient was a large-framed stout man. The whole of his right lower limb up to Poupart's ligament was greatly swollen and œdematous, and large hard swollen areas of a faintly bluish-red colour were observed on the front and inner aspects of the enlarged thigh. The femoral artery in the right groin could not be felt, that in the left

was beating feebly. The whole of the foot and the lower third of the leg were quite gangrenous, being blackish grey, swollen, and œdematous; there was a purplish red discoloration over the calf, and mottling of the skin nearly up to the knee. He still complained of slight weakness and coldness in the left foot, but there were no signs of gangrene there. The veins of the abdominal parietes, especially of the right side, were particularly distended. Skin of trunk moist. Pupils equal. His pulse 88, soft and weak; heart's impulse feeble and ill-defined, no valvular disease, but mitral sounds thought to be not quite clear. Urine clear, acid, 1025. Abdomen and rectum were carefully examined, as it was supposed, from the condition of the limb and the character of the gangrene, that a tumour of some sort must be obstructing both artery and vein as they passed along the pelvis to the groin. This examination afforded no information.

The question of amputation was anxiously discussed, and it was resolved to amputate as soon as time had been allowed for collateral circulation to be established and the gangrene gave any appearance of not further spreading. In the meanwhile the danger of septicæmic infection in such a form of gangrene was considered to be unusually great.

On January 11th the gangrene seemed to have stayed its progress just below the knee. The temperature began to run high, being 102·8°, and on the three following days it sometimes reached 104°.

On the 14th the thigh was amputated in its lower third, the circular method being preferred as it was thought to cause the least division and disturbance of the arteries. A feeble and tiny stream of blood coursed out of the superficial femoral trunk; the main vessel was torsioned. At the time of the operation it was feared that the stump would slough because the muscles were so pale and bloodless, and there was gaseous crepitation felt in the deep tissues of the thigh.

The patient lived eight days after the amputation, the

stump showed superficially no signs of sloughing, but neither did it show any signs of healing; no pulsation returned in the right femoral artery. Slight delirium and diarrhœa set in on the day of the operation, the temperature kept high, subsultus tendinum was marked, the delirium and restlessness increased, the urine became albuminous, the excreta at last were passed unconsciously, vomiting became incessant, the face cyanosed, and death occurred on the afternoon of January 22nd.

The autopsy was made twenty-three hours after death by Dr. Fowler, and the following is the summary of the post-mortem report :—

*General appearance.*—Well nourished. The right thigh had been amputated a little below the junction of the middle and lower third of the femur. There was œdema of the left leg. The veins of the abdomen and lower part of thorax were prominent, and more than the usual number of venous radicles were visible.

Stump : The edges of the flaps were united by sutures; but little reparative action had occurred; there was no actual union at the outer margin of the wound, but the opposed edges here were covered with granulations. On laying open the wound the muscular and intermuscular structures presented a horrible sloughy appearance; the sloughs emitted a most fœtid odour. The hip-joint was intact. There was no infiltration or other change in the integument of the stump.

Heart 12¼ oz. There were a few small soft clots, mostly of post-mortem formation, in the right cavities of the heart with some fluid blood. The (right) valves were normal, the muscular tissue pale and rather soft. The left cavities contained some small post-mortem clots and fluid blood. The valves were competent; there were a few spots of atheroma in the anterior curtain of the mitral valve and also in the first part of the aorta. The muscular tissue was pale and soft, but not obviously fatty.

The left lung showed some emphysema along the anterior edges, and was œdematous in both upper and

lower lobes. The posterior portions of the right lung were completely collapsed and non-crepitant.

*Abdomen.*—The peritoneal cavity contained about two ounces of clear serous fluid. There was a considerable deposit of fat upon the abdominal walls. In the inner margin of the right lobe of the liver close to the round ligament there was a small pale wedge-shaped area, probably an infarction. The liver tissue was pale, soft, and swollen. The liver weighed 71 ounces.

The spleen was large, weighing 9½ ounces, soft and congested. The kidneys were swollen, each weighing 7¼ ounces, the capsule thin and slightly adherent. In the upper margin of the left kidney there was a small, pale, wedge-shaped area much resembling that in the liver. The renal tissue was firm, the cortex showed a slightly granular surface.

Vessels: The aorta, and arteries of the extremities, with the vena cava and corresponding veins, were removed entire and afterwards dissected. They presented the following appearances. The arch of the aorta and the thoracic aorta showed numerous patches of atheroma. Immediately below the point where the aorta passes beneath the diaphragm, and pushing forward the crura of the latter muscle, there was an oval aneurism projecting from the right side of the vessel. It measured externally 2½ inches in its long diameter and was about equal in size to a Tangerine orange. The mouth of the sac, 1¼ inches long and oval in shape, was situated so that its central point nearly coincided with the origin of the superior mesenteric artery. This latter was not, however, involved in the aneurism. From the mouth of the sac some laminated clot was protruding; the sac itself was nearly filled with similar clot. The posterior wall of the sac had been quite destroyed and the aneurism was here limited by the first and second lumbar vertebræ, the bodies of which were deeply eroded—to the extent of half an inch in depth. The intervertebral disc and edges of the bodies were scarcely at all affected. The aorta was blocked at its

bifurcation, the clot extending for nearly an equal distance (about $\frac{5}{8}$ inch) into each common iliac vessel. The central portion of this clot was paler than the upper and lower portions, and appeared slightly laminated. Just above the origin of the right profunda artery a laminated clot (a portion of the clot from the aneurismal sac) was found blocking the vessel; above and below it were some non-laminated coagula. The divided femoral artery of the stump had been twisted, the end was plugged with firm coagula for a distance of two inches. The vena cava was completely blocked by a tapering thrombus from one inch and a half below the margin of the sac of the aneurism. The common iliac veins were both completely blocked; the external iliac and femoral veins were also blocked, the right femoral down to the point of amputation, the left just above the point corresponding to the origin of the profunda artery. A minute branch of the femoral artery was included in a fine ligature which surrounded the cut end of the vein.

(For report of the discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 426.)

# THREE CASES

# (PROGRESSIVE MUSCULAR ATROPHY

## AND INFANTILE PARALYSIS)

ILLUSTRATING THE

## LOCALISATION OF MOTOR CENTRES IN THE BRACHIAL ENLARGEMENT OF THE SPINAL CORD.

BY

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Received December 9th, 1884—Read April 14th, 1885.

THE two cases which I have shown this evening are chiefly important with regard to the localisation of the centres for different groups of muscles of the upper limbs, in the different segments of the brachial enlargement of the spinal cord.

The first case is one of progressive muscular atrophy. The patient is 38 years old, and has been a goldbeater for eighteen years; he attributes his illness to violent muscular exertion, but he has been a lamplighter for the last six years and much exposed to wet and cold. There is no family or previous history of importance. Three years ago he began to have difficulty in extending the

third and fourth fingers of the right hand; he then thinks that the muscles of the right arm began to waste, and he gradually lost power in them.

A year after the onset, the left arm became affected, and he had trouble in supinating the forearm, then in flexing the elbow, and in abducting the arm away from the trunk.

For the last six months the legs have felt weak.

*Present condition.*—In the *right* arm the muscles most affected are, the extensors of the wrist and fingers and thumb, the small muscles of the hand, the interossei, the flexors of the fingers, the biceps, supinator longus, deltoid, rhomboid, serratus magnus, teres major and minor.

The interossei, especially the first, and the muscles of the hand are very much wasted and the extensors of the fingers are more atrophied than the flexors. The fingers are rigidly flexed; he cannot extend or separate, but can just flex them. In the right forearm supination is more difficult than pronation.

The biceps, brachialis anticus and supinator longus are much wasted, and though he has no power to flex the elbow-joint he can just make the two first muscles contract. On the other hand, the triceps is so strong and well developed that he can prevent the elbow-joint being forcibly flexed. The deltoid is very much wasted, specially the middle part, and he is unable to abduct the humerus away from the trunk, although the supraspinatus seems to contract. The pectoralis major acts fairly well, but the sternal better than the clavicular half, and he can with much difficulty flex the shoulder-joint, bringing the arms forwards, though not to the horizontal line. In so doing the scapula projects backwards and away from the trunk, owing to the serratus magnus being much wasted and incapable of advancing the inferior angle of the scapula. The absence of action of the serratus magnus is well seen when the arm is forcibly raised above the horizontal line, and allowed to drop, when it falls at once on removing the

support. In adducting the humerus to the trunk the latissimus dorsi acts fairly well, but not the teres major and minor. He cannot draw the scapula towards the spine by the rhomboid muscle. The upper end of the trapesius acts normally, the rest of the muscle not so well as in health, and in shrugging the shoulders the scapulæ appear to be elevated by the trapezii and not by the levatores anguli scapulæ.

In the *left* arm the changes have not proceeded so far, and there are some important differences. Here the muscles most affected are, biceps, brachialis anticus, supinator longus, deltoid, rhomboid, supra- and infra-spinati, teres major and minor, serratus magnus, extensors of thumb, and to a less degree, the extensors of the fingers. There is not so much wasting of the hand and forearm as on the right. He can flex the fingers and thumb well, and use the small muscles of the ball of the thumb, being able to touch all the tips of his fingers with the thumbs. He can extend the first finger well, but the wrist and other fingers he can only just extend, and the thumb not at all. When the hand is supported the interossei can separate the fingers. The biceps and supinator longus are much wasted and absolutely no voluntary movement can be produced; the inaction of the supinator longus is strikingly seen, in comparison with the extensors of the fingers which he can still throw into action. I may remark that this is just the *opposite* to *lead* paralysis, where the extensors are paralysed and the supinator longus, which is really a flexor of the elbow-joint, escapes; this is important, as lead paralysis is considered by some writers to be due to disease of the cells of the anterior cornua of the cord. On the other hand, the triceps is strong, normal, and well formed. The deltoid, the supra- and infra-spinati are much atrophied and their movements *nil*. The pectoralis major is rather weak in its action and the clavicular more affected than the sternal part, so that he is unable to bring the humerus forward so well as on the right side. In adducting the humerus to the trunk, the

latissimus dorsi acts well and better than on the right, but no action can be seen in the teres minor and major. The serratus magnus does not act at all, but there is not that protrusion of the posterior border of the scapulæ which is so marked on the other side. This is probably due to the upper fibres of the pectoralis major not being strong enough to advance the arm, so that no strain is put upon the upper part of the scapula. The rhomboid is quite powerless to draw the scapula to the spine.

The patient carries his head bent forwards and there is much prominence of the upper dorsal and last cervical spines. He has some difficulty in extending the neck and throwing the head backwards, and then when it is allowed to come forwards again it falls suddenly after it has passed the vertical position; this is due to the weak action of the muscles keeping the head erect—the splenii, complexi, and trachelo-mastoid muscles.

The sterno-mastoids, however, act normally and can turn the head to the right or left.

The pupils are equal, but the right does not act to light, though it does to accommodation.

The left pupil reacts to light and to accommodation.

With regard to the electric reactions of the affected muscles I may state that there is no reaction to the induced faradic current on the *right* side in the interossei, extensors and flexors of fingers and thumb, the deltoid, the rhomboid, the serratus magnus, the spinator longus, and the teres major; of these, the extensors of the fingers, their flexors and the rhomboid only react to a strong constant current and to the positive better than the negative pole; the biceps does react to the faradic current, but in this muscle slight power still exists.

The lower and middle parts of the trapezius react to a rather strong faradic current. In the *left* side the biceps, supinator longus, deltoid, rhomboid, supra-spinatus, infra-spinatus, teres major and minor, and serratus magnus do not react to strong faradic currents; the biceps and supinator longus react to the positive pole of a strong

constant current, as do also the teres major and minor. The pectoralis major reacts to a moderately strong faradic current, which is also required to produce contraction in the middle and lower parts of the trapezius.

The legs, though weak, do not present any marked wasting or loss of power. The patellar tendon reflexes are increased, a condition which I have seen in other cases of muscular atrophy, and especially in one which I published in 'Brain,' (Part xix) where ankle clonus was also produced. This is probably due to sclerosis of the lateral columns secondary to the primary lesion.

The second case which I have brought forward is that of a young man, aged 18, who had infantile paralysis when one year old, following scarlet fever; that is, in less time than two or three months after the fever. He was paralysed in all the limbs, but he recovered more or less with the exception of the left arm, which remained most affected. He states that, as long as he can remember, his right arm has been strong but the left arm weak; he was able to walk from two years old, but the feet have always been more or less contracted.

*Present condition.*—The patient is affected more or less in all his limbs, the left arm being the most disabled. *The right upper limb* below the elbow is normal and quite strong, the muscles of the hand and forearm being very well developed and possessing considerable power.

The triceps, including the long head, is very much wasted and he has not the least power to contract it; he cannot keep the elbow extended nor can he extend the elbow-joint when the forearm is allowed to hang at right angles to the upper arm. The triceps gives no reaction to electric current, either faradic or constant.

The biceps and brachialis anticus and the deltoid are normally developed and act very vigorously. The pectoralis major is very well represented in its upper clavicular part, whilst the lower or sternal part is absolutely gone, not a trace of it being seen, so that the anterior fold of the axilla passes in a line towards the upper end

of the sternum, instead of to the lower. The pectoralis minor cannot be made out. He has very little power in pressing with the right hand on a table when the arms are extended, a movement which is specially due to the action of the lower part of the pectoralis major.

The serratus magnus is not affected, and he can raise the arm high up in the air with considerable strength. The remaining muscles of the scapula are normal, including the infra- and supra- spinati, the teres minor, the subscapularis and teres major, the rhomboid, and the trapezius. The latissimus dorsi is absent and cannot be made to act; when the patient tries to adduct the humerus to the trunk against resistance, the work is done by the muscles of the scapula, which is thereby drawn forwards away from the trunk.

These muscles take the place of the latissimus dorsi in placing the arm behind the back. There is no reaction in the latissimus to faradic or constant electric currents.

In rotating outwards the humerus by the teres minor and infra-spinatus, the scapula is drawn forwards, the rhomboid not having sufficient power to keep it fixed.

The *left arm* is more affected than the right, and is generally wasted, the circumference of the right forearm being eight and a half inches, whilst the left is only seven and a quarter.

The muscles of the ball of the thumb and the interossei are not wasted, and he has good movement in them. The extensor aspect of the forearm is wasted, and he can only just extend the fingers and wrist to the horizontal line; in so doing the wrist becomes flexed, while the fingers are hyper-extended. He has no power to extend the thumb. These extensors require strong faradic current for their contraction. In the upper arm, which is considerably wasted, the biceps and supinator longus act fairly well, but not so strongly as on the right side. The triceps is very much wasted and no contraction can be produced either by the will or by strong faradic or constant currents, with the exception of its long head, which

reacts to a strong faradic and to the positive pole of a strong constant current. The deltoid is also a good deal wasted, but the patient is just able to abduct the arm to the horizontal position.

The serratus magnus is somewhat affected, so that he cannot raise the arm further than the horizontal line, unless the elbow be flexed, thereby reducing the weight of the arm, but this inability may be also due to the weak action of the deltoid. In adducting the humerus to the trunk, no action is seen of the latissimus dorsi or the teres major or minor. What action there is appears to be done by the posterior part of the deltoid, which also has slight power in rotating the hanging humerus outwards. The patient cannot effect any rotation inwards, the special rotators, both in and out, being very much wasted and quite powerless.

The pectoralis major is much wasted, especially the sternal part, of which a thin band only is visible, so that he is unable to keep the left hand touching the right shoulder. The supra- and infra-spinati are very much wasted and powerless. The rhomboid is incapable of drawing back the scapula to the spine, and although the upper fibres of the trapezius act normally the rest of the muscle acts feebly.

In the lower limbs the feet are much altered in shape, the toes being hyper-extended whilst the arch of the foot is very much increased, the length of the feet being thus much shortened. This alteration seems to be due chiefly to the non-action of the interossei, and is similar to the claw shape assumed by the hand when the interossei are affected. The tendo Achillis does not appear to be contracted.

The patient can flex the toes of both feet, and slightly flex upwards the right ankle, but not the left. He cannot evert either foot by means of the peronei, and can only just invert both ankles by the tibialis anticus, the right better than the left. The electric examination shows no reaction in the peronei of the left leg to strong

faradisation and only slight contraction to a strong constant current. The tibialis anticus of the right leg reacts very slightly to a strong faradic current, whilst that in the left reacts only to a strong constant current.

There is no patellar tendon reflex in either leg, which is in marked contrast to the condition in the other case.

With regard to the electric reactions in this case, there is no reaction to a strong faradic or constant current in the *right arm*, of the triceps, pectoralis minor, or latissimus dorsi; the clavicular part of the pectoralis major reacts normally to faradisation, while the sternal half, being absent, cannot of course be tested; the serratus magnus requires a faradic current rather stronger than normal to make it contract.

In the *left arm* there is no reaction to strong currents in the triceps (with the exception of the long head which contracts to a strong faradic, and to the positive pole of a strong constant current), the latissimus dorsi, the supra- and the infra-spinatus, the teres major and minor, or the rhomboid. The clavicular part of the pectoralis major reacts normally, but the sternal part only to a strong faradic current. The extensors of the fingers, the supinator longus, biceps and serratus magnus react to strong faradic currents, as does the deltoid, but the anterior and posterior parts of this muscle react rather better than the middle part; this is of some importance as the deltoid may be looked upon as composed of three muscles, the anterior part of which acts in conjunction with the clavicular part of the pectoralis major, which in this case acts fairly well.

The two cases which have been brought forward may be both considered together, for although one is a case of progressive muscular atrophy and the other is one of infantile paralysis, they are both due to a similar cause, viz. a lesion affecting the cells of the anterior cornua of the spinal cord, and especially of the brachial enlargement. With regard to progressive muscular atrophy it has been considered by Friedreich that the disease lies primarily

in the muscles themselves, but at present most writers consider that the cells of the anterior cornua are the seat of disease.

My object in bringing forward these cases is to prove clinically the correctness of the experiments of Professors Ferrier and Yeo on monkeys. These observers divided the several motor roots of the brachial and lumbar plexuses and on stimulating the peripheral ends various groups and combinations of muscles were put into action. I have drawn up a list of these muscles taken from Ferrier's paper in the 'Proceedings of the Royal Society,' 1881, and also from a paper of his in 'Brain,' Parts 14 and 15; and parallel with this list I have given lists of the muscles affected in my two cases. The condition of the two arms is given separately in each case, so that it can be seen at a glance how each is affected.

There has always been a difficulty in explaining why in diseases like progressive muscular atrophy and infantile paralysis certain muscles should be picked out and others left. The groups of muscles affected do not correspond to the nerve supply of any one peripheral nerve; and the extensors as a whole do not suffer more than the flexors, although they are generally the weaker muscles; for while the extensors of the wrist and fingers are usually the first to suffer, the triceps has always been remarkable for its immunity from attack, and has frequently been noticed as the last to be affected.

On looking at these two cases, I think it will be observed that broadly the one is the counterpart of the other; I do not mean absolutely so, for it is not easy to find hard-and-fast stereotyped limits to clinical cases. Comparing the muscles affected in each case, it will be seen that in that of progressive muscular atrophy they correspond to the muscles assigned by Ferrier and Yeo to the upper and lower ends of the brachial enlargement, whilst the case of infantile paralysis corresponds to the muscles governed by the middle of that enlargement. Although the experiments of Ferrier and Yeo were made on the motor nerves,

the groups of muscles assigned to the different roots correspond also to the anterior cornual cells supplying them.

On looking at the list of muscles affected in the case of progressive muscular atrophy it will be seen that there are some differences between the two arms.

In the right arm the muscles innervated by the fourth and fifth cervical, according to Ferrier and Yeo, are all affected, with the exception in the fourth cervical of the supra- and infra-spinati muscles which have escaped, so that, excluding these two muscles and the diaphragm—which has the nuclear origin of the phrenic in the medulla and not in the cervical cord—not a single muscle has escaped. On looking at the sixth cervical group, the pronators of the wrist are slightly affected, but the serratus magnus is the only muscle seriously damaged, and this is also supplied by the affected zone of the fifth cervical. In the seventh cervical, all the muscles have escaped except the flexors of the wrist and fingers and the teres major; and it is interesting to note that the muscles which are supplied by, and only by, the sixth and seventh cervical have escaped; this is in striking contrast to the case of infantile paralysis, where the same muscles are the only ones which are paralysed. The eighth cervical zone has all its muscles affected excepting the long head of the triceps and the pectoralis major, about which Ferrier has some doubt as to its proper position here. The first dorsal, representing the hand muscles, is entirely damaged.

When we examine the left arm we find that the disease, while following the general course of the other arm, has shifted its position a little higher up, so that we see, contrary to what is usual, that the small muscles of the hand supplied by the first dorsal have escaped, and the disease does not seriously begin till we reach the fifth cervical zone.

The pectoralis major is affected, but in the upper or clavicular part more than the lower or sternal half—in contradistinction to what is found in the other patient—

and I certainly think that the pectoralis major may have its two halves assigned to different parts of the cord, the clavicular half going along with the anterior fibres of the deltoid, of which it is a continuation, whilst the sternal half is more associated with the triceps group. This grouping is also illustrated by physiological action, for in pressing the two hands together the clavicular part of the pectoralis major, the anterior fibres of the deltoid and the biceps are associated, whilst in pressing the hands downwards, against a table, the sternal part of the pectoralis major and the triceps act together, the biceps not being used in this movement, which may be illustrated by pressing on the table to assist in rising from a chair. In the fourth and fifth cervical groups all the muscles are involved, as on the opposite side, but with the exception that the supra- and infra-spinati are also affected, whilst the extensors of the fingers have more power than on the right side.

In the list of the muscles affected in the infantile paralysis case, it will be seen that only the triceps, pectoralis major (sternal half), and latissimus dorsi are absolutely powerless and give no reaction to electric stimulation. Now these muscles, according to Ferrier's list, are innervated *only* by the sixth and seventh cervical roots, and if these are seriously damaged, or rather if the anterior cornua, the segments of the cord containing their nerve-cells, are diseased they become powerless. It is interesting to note that these three muscles are the only ones in the sixth and seventh cervical zones which are supplied by both these nerves; the teres major and subscapularis, both internal rotators, are supplied by the seventh cervical alone, and the pronators by the sixth alone, and these have escaped. I can only suggest that these may perhaps have another nerve supply from a different zone which has not yet been discovered. I think some light may be thrown on this apparent anomaly when we examine the left arm.

In the left arm precisely the same muscles are affected as in the right, but the disease has gone further and involved other zones of the cord. And here we see that

the subscapularis and the teres major, the clavicular portion of the pectoralis major, the rhomboid, the supra- and infra-spinati must be added to the list of muscles severely affected. I think it is interesting to note that the remaining muscles supplied by the sixth and seventh cervical are here invaded, as the disease is more extensive. The pectoralis major is here more affected in the sternal than its clavicular part, following a course similar to the muscle of the right side. The fact of the deltoid being somewhat affected would lead us to place the clavicular part of the pectoralis major along with the deltoid, whilst the sternal part would be assigned to the sixth and seventh dorsal zones.

I confess it is difficult to make the spinati harmonise with the fourth cervical zone.

Besides the above muscles the serratus magnus, deltoid, and extensors of the wrist are somewhat affected, though they possess voluntary movement, and all respond to strong faradisation. They would show a moderate extension of the disease to the fourth and fifth cervical.

It will be seen that the eighth cervical and first dorsal are almost intact, the small muscles of the hand and flexors of the fingers thereby escaping.

It seems probable that in the case of progressive muscular atrophy the disease began with atrophy of the right hand muscles, and so corresponded to the first dorsal segment of the spinal cord; the disease subsequently attacked the upper end of the cord, the fourth and fifth cervical, the middle part of the brachial enlargement not being affected or only very slightly.

It is very extraordinary why the extremities of this enlargement should be most attacked, and why the disease should, so to say, leap over the middle part; perhaps some difference in the blood-supply of the middle and ends of this part of the cord may in future time be discovered. It is of course possible that the disease may begin in the two extremities at the same time, and I think that in the left arm the disease seems to have commenced at the

upper part of the brachial enlargement as shown by the biceps and deltoid being first affected.

In the case of infantile paralysis, the whole of the brachial enlargement was probably entirely affected at first, the ends recovered completely in the right arm, but to a less degree in the left, while the middle has been left permanently paralysed, but why we do not at present know.

A third case which was under the care of Dr. Ferrier at the Queen Square Hospital—whom I have to thank for allowing me to publish it here—is a boy, aged 13, who had infantile paralysis when two and a half years old, affecting the right arm and the legs.

At present the right arm is affected in the triceps group of muscles corresponding to those supplied by the sixth and seventh cervical roots, and therefore corresponds to the second of the cases already described, viz. that of infantile paralysis.

In the right arm he has all the movements of the small muscles of the hand and interossei, and can extend and flex the wrist and fingers, and these muscles react normally to the faradic current. The biceps, brachialis anticus and supinator longus act well but the triceps is very much weaker than in the left arm, and it is important to note that whereas the long head of the triceps (which is placed in the eighth cervical group) reacts to nearly a normal faradic current, the short head reacts very slightly to a strong faradic current and to the positive pole of a rather strong constant current, whereas all parts of the left triceps react to the normal faradic and constant currents. The right deltoid abducts the humerus well, but the sternal half of the pectoralis major is decidedly weaker than the clavicular part, and while this clavicular part acts normally to faradisation, the sternal part requires a much stronger current than does the same part of the muscle on the left side.

The serratus magnus is very weak, and in carrying the right arm forwards to the horizontal line, the posterior

border is seen to protrude very much owing to the weak action of the serratus magnus; the protrusion is most marked just before the horizontal line is reached, but after this is passed and the arm is raised nearly vertical, the protrusion disappears; this may possibly be due to the lower fibres of the serratus being less affected than the upper fibres (?); this muscle reacts very little, if at all, to a strong faradic current. The infra-spinatus and teres minor, the external rotators of the shoulder, act better than the internal rotators, the subscapularis and teres major.

In adducting the right humerus to the trunk against resistance, the latissimus dorsi hardly appears to contract at all, and the scapula is drawn away from the trunk by the teres muscles; the right latissimus acts very slightly to a strong faradic current. The patient has apparently some difficulty in drawing the right scapula towards the spine, but the rhomboids act to nearly a normal faradic current.

In this case it will be seen that the muscles affected belong to the groups innervated by the sixth and seventh cervical roots, the fourth, fifth, and eighth cervical and the first dorsal escaping. In the seventh cervical we have the teres major, latissimus dorsi, subscapularis, pectoralis major (sternal half), and triceps, in fact all the muscles, except the flexor of wrist and fingers, which being also supplied by the eighth cervical escapes. In the sixth cervical, the latissimus dorsi, the pectoralis major, serratus magnus and triceps are affected. It may be said that the serratus magnus ought to have escaped as it is also innervated by the fifth cervical, but it seems probable that this muscle is twofold in its action, and the upper fibres are supplied by the sixth cervical, while the lower fibres inserted into the lower end of the scapula are supplied by the fifth cervical, and this grouping is borne out by the movements produced by Ferrier and Yeo in monkeys.

| FERRIER AND YEO'S LIST. | Progressive muscular atrophy. | | Infantile paralysis. | |
|---|---|---|---|---|
| | Right. | Left. | Right. | Left. |
| 1st *Dorsal.* | | | | |
| Small muscles of hand and interossei | × | | | |
| 8th *Cervical.* | | | | |
| Long flexors | × | | | |
| Ulnar flexors of wrist | × | | | |
| Small muscles of hand | × | | | |
| Extensors of wrists and fingers | × | — | | — |
| Triceps, long head | | | × | — |
| (Pectoralis major ?). | | — | Sternal part | |
| 7th *Cervical.* | | | | |
| Teres major | × | × | | × |
| Latissimus dorsi | | | × | × |
| Subscapularis | | | | × |
| Pectoralis major | | — | Sternal part | × |
| | | | × | |
| Flexors of wrist and fingers (median) | × | | | |
| Triceps | | | × | × |
| 6th *Cervical.* | | | | |
| Latissimus dorsi | | | × | × |
| Pectoralis major | | — | Sternal part | × |
| | | | × | |
| Serratus magnus | × | × | | — |
| Pronators (flexor of wrist ?) | — | | | |
| Triceps | | | × | × |
| 5th *Cervical.* | | | | |
| Deltoid (clavicular part) | × | × | | — |
| Biceps | × | × | | |
| Brachialis anticus | × | × | | |
| Serratus magnus | × | × | | — |
| Supinator longus | × | × | | |
| Extensors of wrist and fingers | × | — | | — |
| 4th *Cervical.* | | | | |
| Deltoid | × | × | | — |
| Rhomboid | × | × | | × |
| Supra-spinatus | | × | | × |
| Infra-spinatus | | × | | × |
| (Teres minor) | × | × | | × |
| Biceps | × | × | | |
| Brachialis anticus | × | × | | |
| Supinator longus | × | × | | |
| Extensors of wrist and fingers | × | — | | — |
| Diaphragm | | | | |

× Severely affected. — Slightly affected.

Ferrier has since found that the above list is one root too high ('Proc. Roy. Soc.,' vol. 35, 1883, p. 229), and the groups of muscles should refer from the

fifth cervical to the second dorsal, so that the group assigned to the fourth cervical really belongs to the fifth, and the fifth to the sixth, &c.

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 430.)

# ON THE PATHOLOGICAL HISTOLOGY

OF THE

# SEMILUNAR AND SUPERIOR CERVICAL SYMPATHETIC GANGLIA.

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WISHING to discover whether or not the sympathetic ganglia were affected in certain diseases, I have been occupying my spare time during the last two years in preparing a number of sections, in all over a hundred and fifty, of the semilunar and superior cervical ganglia, with a view to discovering how far the structure of these bodies might vary within normal limits. Inasmuch as this subject is very cursorily referred to in any text-books, with the exception of one or two Italian ones, I thought the results of my investigations might be of use to some members of our profession.

In order to avoid any bias I adopted the following method of procedure. As soon as the post-mortem examination was made the specimen was put into a bottle which was numbered, whilst the description of the post-mortem was entered in a book against this number. The

sections were in due time cut and a description of the appearances presented was written out; after this was done, by reference to the number of the post-mortem, the disease of which the patient died could be discovered. It will thus be seen that in no case was I aware of the cause of death when I described the microscopical appearances.

Before going any further I would point out that the size of the ganglion, whether superior cervical or semilunar, is of no pathological significance whatever. Thus I have seen the superior cervical vary from a quarter of an inch to more than an inch in length, and in the latter case the width was increased in proportion; again I have noted that the middle cervical, which is usually so small, may be actually larger than the superior. In the case of the semilunar I have always cut the sections so as to get the largest area, and whilst the largest measures an inch and a half by rather over half an inch, the smallest is no larger than the section of a split pea. The size bears no relationship to the cause of death, for in some instances in which I have cut sections of the same ganglion from different cases of the same disease, the size has varied considerably.

As a rule the shape of the superior cervical is that of a spindle, and that of the semilunar, like the thumbnail; but I do not think that any importance should be attributed to differences in shape, for very often the semilunar is of such a shape that it cannot be likened to any object whatever, and I have seen the middle cervical so irregular that it might have been mistaken for a small semilunar. Giovanni[1] practically agrees with what I have said, for although he says that alterations in the quantity of the constituent elements of the ganglia alter their size, he points out, what his tables show, that the variations in volume are too great to be of any service to pathology.

The naked-eye vascularity of the sympathetic system is of no pathological significance, for the vessels are so small

[1] 'Patalogia del Simpatico,' di A. De Giovanni.

that no constant arrangement can be described; thus, sometimes the artery before entering the ganglion will run on it for some little distance, whilst in others it will enter it directly. What I have here pointed out is markedly shown in my preparations; thus, in making the post-mortem on a case of myxœdema, the middle cervical ganglion appeared so vascular that all who were present thought it abnormal. Microscopic examination showed no increased vascularity; it was merely an anatomical accident that the vessels were running some way over the surface before entering the ganglion. The greatest vascular engorgement I have ever seen occurred in a case of diabetes, but this was only to be noted after microscopical examination, not being visible to the naked eye. That apparent hyperæmia is valueless is also seen from the fact that Giovanni records that it existed in pleuro-pneumonia, tubercle, cardiac disease, atheroma, chronic nephritis, typhoid fever, cancer, puerperal peritonitis, hydrophobia, and diphtheria. A condition present in such a variety of diseases cannot have much significance.

I do not think it is possible to say much about œdema of the ganglia, at least as being visible to the naked eye. This is not surprising when we remember their small size, the denseness of their capsule and of the interstitial connective tissue; as in the case of hyperæmia, Giovanni's enormous number of ganglia which he says were œdematous show that either it can be of no importance, or, what I suspect is probably correct, that he has recorded many which were really quite normal as œdematous.

With regard to surrounding fat, the superior cervical ganglion has none, and that around the semilunar varies very much in quantity, but without affecting the internal structure of the organ any more than does that around the kidney or heart. The ganglia are generally of a solid firm consistency. I have not noticed any variations in this respect in the various specimens I have examined.

I have met with no case of adhesion of the ganglia to the surrounding parts.

It will thus be seen that I have come across no example of any external appearance of the ganglia being of any significance whatever either as an indication of obvious disease or of what we may expect to find internally; in fact, I should be inclined to say that the only possible cases in which the external appearance of the ganglia could be of any importance are, firstly, those in which one or more of them were implicated in, or affected secondarily by, some morbid growth such as carcinoma or sarcoma; secondly, those in which an aneurysm, abscess, tumour, or other new formation pressed on them; thirdly, those in which a mechanical injury has affected them; fourthly, those in which some chronic inflammatory or malignant process either spread into them from surrounding parts, or by contraction of the newly-formed fibrous tissue pressed upon them; fifthly, those in which an enlargement was caused by something internal, such as an abscess or a tumour. It will be seen that all the above are theoretical affections of which we have very little knowledge; they are introduced chiefly to show how rare any morbid affections of the ganglia visible to the naked eye must be. The trunk of the sympathetic is more often implicated as is seen in some cases of aneurysm. It is of course conceivable that sometimes the sympathetic should be affected by general conditions such as tubercle and lardaceous disease, but of this I have no experience.

We now come to the microscopic appearances of the ganglia, and the nerve-cells, as they are of so much importance, will first of all occupy our attention.

The typical ganglionic nerve-cell from the sympathetic ganglia is like a nerve-cell from elsewhere. It is large, takes the logwood stain well, is rounded, has one or more processes, and a distinct nucleus and nucleolus. I find that among the cases which I have examined the following are marked as presenting cells which exactly correspond to the above description:—Cancer of bladder, aortic disease, sarcoma of breast, double phthisis, bronchopneumonia, chronic Bright's disease (two cases), cancer of œsophagus,

rupture of intestine, stricture of urethra, diphtheria, and scald. It is especially to be noted that in the two cases of chronic Bright's disease, the description of the cells states that they are exceptionally typical, for Dr. Saundby[1] has written a paper in which he says that in chronic Bright's disease the ganglion cells are abnormal, being pigmented and degenerate. He also refers to a paper by Drs. Da Costa and Longstreth, in the 'American Journal of Medical Science' for July, 1880, and states that these authors go so far as to attempt to explain the phenomena of Bright's disease by these changes in the cells. My experience is that patients dying of this malady are not more liable than any others to have abnormal cells in their sympathetic ganglia. The two cases of diphtheria and scald were children, the cells were exactly similar to those from adults.

I do not think any importance can be attached to the number of the cells found; for, in the first place they are so irregularly scattered about in the ganglia that we can never be sure that one section shows them in their maximum number, sometimes the section happens to be taken through a part which consists of little else than nerve-fibres and some fibrous tissue; in the second place, ganglion cells may so often be found in what is to the naked eye sympathetic nerve trunk, that even if we were inclined to think the ganglion contained but few cells, it is quite possible that this deficiency would be compensated for by a large number of cells being present in the nerve trunk. It is far more common to see sympathetic ganglion cells without processes than with them, the absence does not appear to be connected with any particular disease and is I think of no pathological value.

Often no nucleus or nucleolus can be seen in an otherwise healthy cell; there is no present evidence to show that the presence or absence of the nucleus is of any more importance than the presence or absence of processes.

The typical nerve-cell from either of the sympathetic

[1] 'Brit. Med. Journal,' Jan. 13th, 1883.

ganglia now under consideration is larger and more rounded than an anterior cornu cell from the cervical spinal cord. Its difference in shape is clearly due to its not sending off so many processes. Sometimes the cells may be smaller, even half the size, and still, as far as one can judge, they are capable of functional activity. In specimens from the following cases the cells appeared to be of a normal size: anthrax, cancer of bladder, aortic disease, sarcoma of breast, double phthisis, broncho-pneumonia, cirrhosis of the liver complicated with granular kidney, another case of granular kidney (concerning these specimens I have made the note that I have seldom seen better examples of normal cells), rupture of the intestine with peritonitis, stricture of urethra, phthisis, tumour of brain, another case of granular kidney where it is likewise remarked that the cells are remarkably good, diphtheria, scald.

In all the above the size of the cells is normal over all or nearly all the specimens. In some other cases they are small in one part and large in another. It must also be borne in mind that the size of a cell will depend somewhat on the direction of the section. When a cell is small, that diminution in size is nearly always due to pigmentary degeneration, which we will consider presently; some cells are, however, pigmented without any alteration in size. In cases dying of the following diseases the cells were diminished sufficiently in size for the diminution to be noteworthy: diabetes (four cases), idiopathic anæmia (two cases), abdominal aneurism (superior cervical ganglion, the cells are reduced to a minute mass with no resemblance to the original), broncho-pneumonia, sarcoma of pelvis, gangrene of foot from atheroma, malignant disease of the bladder, aneurism of abdominal aorta (semilunar ganglion), aortic disease, myxœdema, general malignant disease.

On contrasting this list with that in which the cells are not diminished in size, it will be seen that the second enumeration contains many more wasting diseases than

did the first: thus there are four cases of diabetes and three of malignant disease. So we may say that as a general rule wasting of cells is most marked in wasting diseases, but that this is subject to many variations.

We now come to the degenerations of the cells, of these the only one of any importance is the pigmentary. This may or may not be accompanied by diminution in size. In all the specimens above mentioned, as showing a decrease in size, some or other of the cells were pigmented; in slides taken from the following diseases the cells were pigmented although not diminished in size: anthrax, cancer of the bladder (two cases), aortic disease, sarcoma of breast, phthisis (two cases), chronic Bright's disease (three cases), purpura, broncho-pneumonia, cancer of œsophagus, abdominal aneurism, rupture of the intestine, tumour of the brain.

Pigmentation occurs in specimens taken from people dying of almost any disease, and in consequence of its almost universal presence, in greater or less quantity, in the cells of almost all specimens of sympathetic ganglia one is compelled to look upon it as of no pathological importance. The only thing to be noted is that it is entirely absent in the two cases in which the ganglia of children were subject to examination. It is just possible that the interpretation of this almost universal pigmentation is that it is connected with the smaller ailments from which none of us are free. This might perhaps apply to the semilunar ganglia with more force than the cervical, but both seem to be with equal frequency pigmented. Reference to Giovanni's work will show that age has very little to do with the quantity of pigmentation, so that perhaps it is no more than an accident that the two cases of children which I examined show no pigment.

This pigmentary degeneration usually occurs as small, roundish, bright yellow masses in the nerve-cell; frequently it may be observed that whilst part of the cell has undergone pigmentary degeneration part is quite free and well stained with logwood, which agent has no effect

on the degenerate part. The intensity of the yellow colour is liable to fade if the specimen has been kept long. If the change be extreme the cells are represented by little masses of yellow pigment shrunk away from the capsule, bearing but little resemblance to the original cell, which thus sometimes looks like the section of a vein with a little blood-pigment in it. I do not think that this condition can be shown to be associated with any particular condition of the blood-vessels or surrounding tissues.

Sometimes the cell has a fine granular appearance. I am unable to connect this with any particular disease. Considering that the pigment tends to disappear the longer the specimen is kept, I am inclined to think that these granules may be pigmentary ones from which the colouring matter has become dissolved out.

Often the cell presents a vague, ill-defined outline, so that it is almost impossible to define its margin. It is more common for this condition of border to exist at one part of the cell only, it may, however, exist all over. It is frequently coexistent with absence of the nucleus; in such cases the contrast between a healthy cell with a well-defined nucleus and the vague non-nucleated misshapen one is very striking. Like the other variations of the cell this vague condition cannot, as far as I know, be connected with any particular disease. Although I have occasionally seen what I have taken to be fat granules present in a cell, I certainly should not think fatty degeneration to be so common as one would infer from Giovanni's statements. Of other degenerations of nerve-cells in sympathetic ganglia I have no experience. In some of the best specimens the nucleus does not stain at all, but stands out as a bright colourless spot with a dark nucleolus in the centre, in others the nucleus stains dark.

From a study of the cells one is driven to the conclusion that in the present state of our knowledge their varying appearance cannot be said to be of any significance; either the variations may be very great and the cell be still normal, or, in at least four fifths of the cases examined,

the cells were abnormal; and if the latter hypothesis be true we are still unable to connect the abnormal condition of cell with any particular disease or symptoms.

Taking next the nerve-fibres they are quickly disposed of. It is often difficult to distinguish them from the connective-tissue fibres, but it is to be remembered that they usually run in much more regular parallel bundles than the latter, these parallel bundles when cut contract up and give an appearance very like that of geological strata that have undergone a little upheaval. Although the majority of the fibres are grey, a few white ones may be occasionally seen. I have never seen pigmentary degeneration of nerve-fibres, nor indeed have I seen any changes in them of which I could speak with much confidence. When it is remembered that they are irregularly mixed up with the connective tissue of the ganglion the difficulty of distinguishing any sclerosis of the nerve-fibres in the ganglion will at once be apparent.

With regard to the fibrous stroma of sympathetic ganglia one can never give an opinion as to whether the quantity is abnormal or not, for not only does it vary very much in different parts of the same ganglion, but the amount present varies much in different ganglia, a fact which is not surprising when we remember how they themselves vary in size. Then, again, the fibrous tissue may appear to be more than it really is from the section having been so cut as to include a great deal of connective-tissue fibre, for that is more abundant at the surfaces than elsewhere owing to its forming a fibrous sheath for the ganglion. In some sections which I have examined the space which generally exists between the nerve-cell and its capsule has disappeared, and this would seem to be due to the connective-tissue stroma pressing down the capsule tightly on the cell. This state of things was observable in sections taken from cases of cancer of the bladder, glioma of the brain, myxœdema, and chronic Bright's disease. Whether or not this is to be regarded as abnormal and can be looked upon as evidence of in-

creased interstitial tissue, is, I think, very doubtful, seeing that it occurs in such widely different diseases in which there is no reason to suspect any sympathetic change. It is, however, the only evidence, slight though it be, of increased connective tissue, that I have found. Then, again, as we have been unable to decide whether the specimen shall be considered abnormal when the cells have undergone pigmentary degeneration, we ought to hesitate before we say too much about slight pressure on them. Perhaps of all the specimens I have examined one of the semilunar ganglion from a case of abdominal aneurism shows most fibrous tissue, and in this one the nerve-cells are much atrophied, although the capsule is not pressed in. This is mentioned because of the possibility that it may be connected with pressure on nerves by the aneurism. Sometimes the capsule around the cell stains well and shows numerous nuclei, at other times but little of it is to be seen.

Next comes the consideration of the vessels. In the first place let it be borne in mind that vessels are much more numerous in the superior cervical than in the semilunar ganglion. As might be expected, considering their varying shape, I have not been able to discover any constant arrangement of the vessels in the semilunar, whilst in the superior cervical the normal arrangement is for a good-sized vessel to enter at the side, as the central artery of the retina enters the optic nerve, and then to run vertically down the centre of the ganglion in its long axis; this artery gives off several branches and is accompanied by a vein and a good quantity of connective tissue in the form of a sheath. If the small vessels be very evident it may be taken as evidence of congestion. In one diabetic specimen they were so numerous and seemed so dilated that although there was no blood in them, one was forced to the conclusion that there was congestion, and that the blood had dropped out in the preparation of the specimen. The most extreme congestion I have seen was from other cases of diabetes; here

the vessels were very numerous and all crammed with blood-corpuscles. Diabetes is the only disease in which the congestion has been sufficient to be noteworthy. I have not been so fortunate in finding hæmorrhage as Giovanni, for I have never seen it in the substance of the ganglion and only once in its sheath; this occurred in sections of the superior cervical ganglion in a case of abdominal aneurism, but not the same one as I have just mentioned as having the increased fibrous tissue in the semilunar ganglion. In some examples of chronic Bright's disease the minute arteries have their walls thickened, but, in this disease, I have generally found that, with this exception, the ganglion is healthy.

We have now to treat of inflammation of the ganglion. Only acute inflammation calls for consideration, for we have seen how difficult it is to pronounce on chronic hyperplasia of the fibrous tissue. The characteristic of acute inflammation is the presence of innumerable small cells quite obscuring the section, so that the component nerve-cells can with difficulty be distinguished; this may be accompanied by congestion. Very many of these cells are undoubtedly white blood-corpuscles, for in some sections they may be seen in the act of passing out of the vessels; but it is very probable that some are due to a proliferation of the cells of the connective tissue, for there is an undoubted increase of the elongated nuclei of the connective tissue which forms a sheath for the bundles of nerve-fibres. I have recorded elsewhere[1] that I have found this condition of inflammation in diabetes; I have also seen it to a slight degree in one case of each of aortic disease, tumour of the brain, and in a child who died from the effects of a scald, but excepting in diabetes it has not been present in an extreme degree save in a case of purpura hæmorrhagica. Considering the lessons we have learnt as to the great variations which may be present, yet the ganglion must not be set aside as abnormal, I should not be disposed to make much of the three cases in which the inflammation was slight,

[1] 'Path. Trans.,' vol. xxxvi.

perhaps it was connected with some trivial ailment. Before discussing the case of purpura hæmorrhagica it would be better to have more examples, this one shows, however, undoubted extreme inflammation. Cases of idiopathic anæmia which I have examined do not show any inflammation. The last condition I have to mention is that in two cases of sections of the semilunar ganglion from children I have found little masses of lymphoid tissue in the ganglion. These masses are not diffuse but each has a distinct capsule. It would seem as though one of the very numerous small lymphatic glands near to the semilunar ganglia has got inside instead of outside of it. As a result of the examination of many sections of semilunar and superior cervical ganglia I may, I think, say that the only lesion which can be positively said to be abnormal is the acute inflammatory condition just described, in which the section is crowded with small cells: therefore the only diseases in which I have found the ganglia undoubtedly deviating from the normal are diabetes and purpura hæmorrhagica. In chronic Bright's disease the vessels in the ganglia are thickened, otherwise the ganglion is healthy.

This is I feel a poor result for so much work as I have gone through, but that is one reason why I have brought this paper before the Society, so that others may be saved the trouble of going over the same ground that I have.

The fullest descriptions of the pathological histology of the sympathetic with which I am acquainted are given by A. De Giovanni, 'Patologia del Simpatico,' and by Foa in the 'Rivista Clinica di Bologna,' 1874, p. 206.

Polaillon[1] in an article which is not of much interest from a pathological point of view, gives a very good historical summary of the knowledge of the normal structure of sympathetic ganglia up to 1866. He says that the granules so frequently seen in the nerve-cells are due to post-mortem affection of the proper substance of the cell. There is, I think, however, no doubt that this is not so, for if it were it should be present in all the ganglia

[1] 'Journal de l'Anatomie et de la Physiologie,' 1866.

that I have, for they were all prepared the same way, and also it should be present in other nerve-cells such as those of the spinal cord. Virchow also thinks that these changes in the nerve-cells are ante mortem; he describes them in fevers and old age.

Dickson suggests that in locomotor ataxy the gastric cases may be due to the affection of the semilunar ganglia. This may or may not be so, but the evidence he adduces is not proof, for he says Dr. Clarke has found in some cases great pigmentation and that these observations have been fully confirmed by those of MM. Poincaré and Henry Bonnet.[1] I have already shown that pigmentation is of too universal an occurrence to be of any importance in explaining any rare malady such as the gastric crises of locomotor ataxy. The French authors just mentioned found changes in general paralysis of the insane. Dr. Savage[2] who has had a large experience, has discovered no changes in the sympathetic which can be constantly associated with insanity.[3]

[1] 'Annales Médico-Psychologiques,' 4me série, Tome 12me, 1868.

[2] 'Insanity, and allied neuroses,' Lond., 1884.

[3] Whilst this paper was passing through the press, Dr. Long Fox published a book on 'The Influence of the Sympathetic on Disease.'

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 436.)

## DESCRIPTION OF PLATE III.

(On the Pathological Histology of the Semilunar and Superior Cervical Sympathetic Ganglia, by W. HALE WHITE, M.D.)

FIG. 1.—Section of a semilunar ganglion from a case of anthrax. Normal. × 250 diameters.

FIG. 2.—Section of a semilunar ganglion from a case of carcinoma of the breast. Normal. Shows processes well. × 250 diameters.

FIG. 3.—Section of a superior cervical ganglion from a case of abdominal aneurism. Shows the extreme atrophy of the cells, which are reduced to mere masses of pigment in the centre of their capsules. × 250 diameters.

FIG. 4.—Section of a superior cervical ganglion from a case of atheroma of the arteries and gangrene of the leg. Shows extreme granular pigmentation of cells. × 300 diameters.

FIG. 5.—Section of a semilunar ganglion from a case of purpura hæmorrhagica, showing the abundance of leucocytes and proliferation of nuclei. × 300 diameters.

FIG. 6.—Shows the contrast in size between the largest and smallest semilunar ganglia met with.

FIG. 7.—Shows the contrast in size between the largest and smallest superior cervical ganglia met with.

Fig 1.

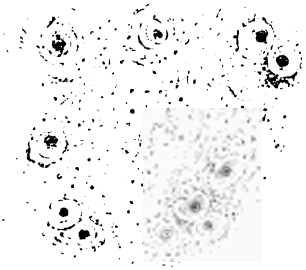

Fig 2

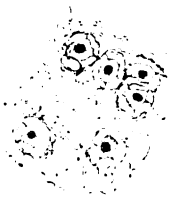

Fig 3

Fig 4

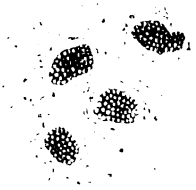

Fig 5

Fig 6

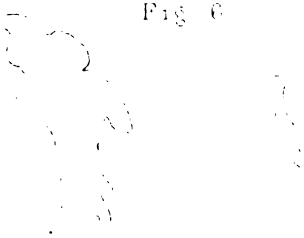

Fig 7

# NOTES

ON SO-CALLED

# NON-OVARIAN DERMOID ABDOMINAL TUMOURS.

BY

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THE object of this contribution is to show that many dermoid cysts of the abdomen that have been described as non-ovarian, are really ovarian cysts that have become separated from their pedicles. This is especially the case with regard to cysts of the great omentum. Not that it can be denied that dermoid abdominal cysts may originate independently of the ovary, or that cysts of the great omentum may be non-ovarian, but growths of the former class are very rare, whilst evidence is wanting that primary cysts of the great omentum are ever dermoid.

In October, 1884, a married woman, aged 33, was admitted into the Samaritan Hospital. She had always enjoyed good health. Six years before her admission she noticed something moving about in her abdomen—a lump lying rather to the left of the umbilicus. Since that date she had been pregnant, and the lump had shifted to the right side. The lower part of the abdomen was occupied,

on the right side, by an obscurely fluctuating tumour that extended towards, but not deeply into, the right flank; it passed only an inch or two beyond the middle line to the left, and did not stretch the abdominal walls between the umbilicus and pubes after the manner of ovarian tumours. There was tympanitic resonance over the tumour, especially towards the right side. The pelvic viscera were quite free from any sign of disease or implication with the tumour.

On October 27th Dr. Bantock operated upon the patient. An incision was made along the outer border of the right rectus abdominis muscle. The surface of the tumour was exposed; it appeared to be smooth, pale, and slightly shiny. On tapping with a Dieulafoy's aspirator, a pint of fluid resembling pus was drawn off. The tumour was then found to be intimately connected with the great omentum and strongly adherent to the ascending mesocolon. It was not deemed judicious to lay it open before removal, lest the contents, which appeared to be purulent, should escape into the peritoneal cavity. After the tumour had been cut away, the right ovary was drawn up out of the pelvis and found to be quite healthy. The left was not searched for, as there were no direct grounds for suspicion at the time that the tumour might be ovarian. On examining the tumour after the operation it was found to be stuffed with hair, lubricated with sebaceous material. There was no trace of either tube or pedicle. On the inner wall were some plates of bone and a tooth of very anomalous form, somewhat resembling a bicuspid, but tapering almost to a point at its edge.

Both the operator and myself more than suspected after examination of this tumour that it was a cyst of the left ovary. Complete separation of the pedicle of a dermoid cyst is not unknown, and in such cases it is generally to the omentum that the cyst adheres. I have described and figured elsewhere[1] a case under the care of Mr. Thornton

[1] 'Clinical and Pathological Observations on Tumours of the Ovary, Fallopian Tube and Broad Ligament.' Figs. 27, 28.

where an abdominal tumour had existed for seven years, during which period the patient gave birth to four children. At the operation a dermoid cyst was found, closely adherent to the omentum. The left ovary was absent, a short, firm tag of tissue hanging from the omentum close to the Fallopian tube. It is very probable that the former case was of the same kind as the latter, and that many other instances of alleged non-ovarian dermoid-cysts should properly be placed under the same category. In the first of the above cases, had the operation been performed many years ago, the ovarian nature of the cyst might never have been suspected, and had the patient recovered, no record of the state of the pelvic organs would have been obtainable. It would probably have been described as a dermoid cyst of the omentum.

A week after the operation in this case, I assisted Dr. Bantock at an ovariotomy where a curious condition of the ovary was detected, which may throw light on an unexpected manner in which dermoid and other ovarian cysts become adherent to the omentum. The patient, who had been under the care of Dr. Amand Routh, was a woman aged 42, the mother of several children. On opening the abdominal cavity, a small, almost unilocular papillomatous cystic tumour of the right ovary was discovered and removed. On examining the great omentum, which was thin and healthy, a soft white body was found adherent to its posterior aspect towards its right free border, almost at the level of the umbilicus. A pedicle about four inches in length was traced to the left angle of the uterus and consisted of a dark red, cylindrical body, which proved to be the Fallopian tube; of a long fibrous cord, the utero-ovarian ligament much hypertrophied; and of some long vessels of small calibre, the ovarian artery and veins. The soft, white body proved to be the left ovary. It contained one dilated follicle, about half an inch in diameter, and was removed, together with the portion of omentum to which it adhered.

It is not easy to understand how this ovary reached

its position behind the omentum, which did not descend unusually low. I know of no previously recorded case where an ovary, not enlarged, had become displaced in this manner. The tumour of the opposite ovary could hardly have dragged it out of the pelvis, for it was not adherent to that tumour. It is also difficult to believe that the tumour could have pushed the ovary up as it enlarged; if a cystic ovary can push up the opposite unenlarged ovary it is singular that this condition is not more frequently seen. This latter argument applies with yet greater force to the theory that pregnancy might have drawn up the left ovary. We do not find ovaries drawn up in this manner in women who have borne children. It is probable that in the case under consideration the ovarian ligament and tube were congenitally of abnormal length, and that the ovary had become displaced from some peculiar habitual position of the patient.

In some previously recorded examples of cysts adherent to the omentum, it is possible that the ovary had become adherent before developing cystic disease. The stretched tube and ovarian ligament would in such a case be very prone to atrophy, and the tumour would then of necessity receive its vascular supply from the omentum. I do not, however, believe that a tumour can increase after torsion of its pedicle. Freund has rightly questioned the possibility of adhesions supplying sufficient blood to a non-malignant tumour, so as to enable it to continue to enlarge when separated from its original channels of supply.[1] In these adhesions, except when the tumour is malignant, the veins greatly exceed the arteries in size and number. Occasional increase in the bulk of the tumour is due to congestion or intracystic hæmorrhage.

I now come to the main argument suggested by the above cases, which is, that many cases of dermoid cyst of the abdomen recorded as non-ovarian were probably ovarian dermoid cysts that had become adherent to the

[1] "Extra-uterine Gestation," 'Edinburgh Medical Journal,' September November, and December, 1883.

omentum or to other structures, and separated from their pedicles. This cannot always be the case, for dermoid abdominal tumours have been met with in males, and none of the fœtal relics of the female type persistent in the male could possibly adhere to the omentum and break off their pedicles, even if they did become subject to dermoid cystic disease. Thus in Dr. Ord's case[1] the dermoid cyst found between the bladder and rectum of a man, might and probably did arise from a fœtal relic, but it had no pedicle and could not have become detached from its pelvic connections. Turning to Lebert's original article on dermoid cysts,[2] where clinical details are more complete than in his later observations on the subject in the 'Traité d'Anatomie Pathologique,' I find that there is strong reason to doubt that all of the cases he describes as non-ovarian had no connection with the ovary.

The cases of dermoid cysts in the testis, brain, facial structures and thorax, as well as of similar cysts in the liver, stomach, diaphragm, &c., in males may be set aside at present. One case of dermoid cyst in the liver of a woman might have been an instance of adhesion of a dermoid ovarian cyst to that organ; still the hepatic origin of the cyst is more probable. Several cases of dermoid cyst of the uterus are described by Lebert. The first was originally noted by Fabricius Hildanus, but Lebert states that there was some doubt as to the precise seat of the tumour, anatomical details being very incompletely recorded. The same authority mentions cases of dermoid tumour of the uterus described by Samson, Birch, Tyson, and Osiander, but they all appear to be based on the evidence of hair and teeth passed during or after labour, and it is most probable that all, including Hildanus's, were instances of ovarian growths acquiring connections with the uterus and upper part of the vagina, with subsequent communication between their cavities.

[1] "An Account of a large Dermoid Cyst found in the Abdomen of a Man," 'Med.-Chir. Trans.,' vol. lxiii, 1880.

[2] "Des Kystes Dermoïdes et de l'Hétérotopie Plastique en Général," 'Gazette Médicale de Paris,' 1852.

Turning to three cases quoted by Lebert as examples of non-ovarian dermoid cysts of the abdomen in women, where the tumour was attached to the peritoneum, the evidence that they were truly non-ovarian is generally defective. I put aside several other cases where the dermoid cyst was developed between the uterus and rectum, not only because the origin of the tumour in such cases is obscure, but chiefly because these cases do not bear directly on the subject of the present communication. I find that Ruysch has recorded a case of dermoid cyst of the great omentum, but there is no evidence that the cyst was non-ovarian. Andral describes a case of dermoid cyst growing between the layers of the mesentery of a negress, but makes no mention of the state of the ovaries. Peritoneal relations become so confused, in many cases of abdominal tumour, as to deceive experienced anatomists. Schutzer has described a case of dermoid cyst of the mesentery in a girl aged 15, but this appeared to be an instance of included fœtation, as parts of a skeleton were found. Scortinagna notes a case of dermoid cyst of the peritoneum discharging for five years through an abdominal fistula, but there is no mention of the ovaries. Besides, before the possibility of separation of a pedicle was recognised, the stump of the pedicle might have been taken for an atrophied ovary. A case of dentigerous cyst of the omentum in a young girl, described by Laflize and Bucher, occurred in 1763, and here again the state of the pelvic viscera is unnoticed.

The last case of this kind in Lebert's series is remarkable. A woman over forty years of age had been for several years under the treatment of Dr. Roux, in the South of France. A large abdominal tumour extended from the stomach to the bladder. The patient died suddenly. Beneath the great curvature of the stomach a large cyst was found. On each side a "broad peritoneal fold" fixed it to the small intestines. It was separated from the bladder by a smaller cyst, the size and shape of a pear. The tumours were dermoid. No mention is made of the

ovaries, nor of the great omentum; indeed, it seems probable that the large cyst lay in the omentum. The necropsy was made "by a doctor from St. Maximin," and the account of the examination was written and sent to Dr. Roux by a veterinary surgeon who was present on the occasion.

Such are the cases recorded by Lebert, whom I find constantly quoted as an authority in contemporary literature on the subject of dermoid cysts. It is to be remembered that of late years, since ovarian pathology has been more extensively studied, and since surgeons have made a rule of carefully searching the pelvic viscera when performing abdominal section, we have not heard of non-ovarian abdominal dermoid cysts in women. From the evidence of cases collected by Lebert, as compared with the specimens[1] upon which this paper is based, I am inclined to think, as I have already declared, firstly, that non-ovarian dermoid tumours within the abdominal cavity are extremely rare, but do occur, the chief proof being their occasional presence in male patients; and secondly, that the great majority of dermoid cysts described as non-ovarian are examples of tumours that were of ovarian origin, but had become adherent to other structures and then had separated from their pedicles. Dermoid cysts of the great omentum are especially doubtful in this respect, and no museum specimen of such a disease can be safely held up as an example of a dermoid tumour springing primarily from the omentum, unless there be very clear evidence that the pelvic viscera have been carefully searched, and both ovaries accounted for.

[1] These specimens are preserved in the Museum of the Royal College of Surgeons of England, Pathological Series, Subseries Diseases of the Ovaries.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 433.)

# CASE OF CEREBRAL TUMOUR.

BY

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THE SURGICAL TREATMENT

BY

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SURGEON TO UNIVERSITY COLLEGE HOSPITAL.

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THE chief features of interest in the case, to which the attention of the Society is directed, are, that during life the existence of a tumour was diagnosed in the brain, and its situation localised, entirely by the signs and symptoms exhibited, without any external manifestations on the surface of the skull. This growth was removed without any immediate injurious effects on the intelligence and general condition of the patient. Although he died four weeks after the operation, the fatal termination was due, not to any special effects on the nervous centres, but to a secondary surgical complication. The case, moreover, teaches some important physiological, pathological, and clinical lessons, and suggests practical reflections which may prove useful to future medicine and surgery.

*History.*—The patient was a farmer, æt. 25, who applied for advice to the Hospital for Epilepsy and Paralysis, Regent's Park, on November 3rd, 1884. His chief complaint was paralysis of the left hand and arm, which incapacitated him from work. He stated that his family history was unimportant, that he had always been temperate and in robust health, and that he never had suffered from syphilis or a day's illness of any kind in his life. About four years ago, while in Canada, a piece of timber fell from a house, struck him on the left side of the head and knocked him down. He thinks he lost consciousness for a few moments, after which he so far recovered as to be able to resume his work. On the following day he was quite well. With the exception of occasional slight headaches he afterwards remained in good health for a year, at the end of which time he first began to experience a feeling of twitching in the left side of his mouth and tongue. This soon developed into attacks of a paroxysmal character, which gradually became more pronounced and frequent, and continued to occur at irregular intervals. Some months afterwards he had a "fit" which began with a peculiar feeling in the left side of the face and tongue, and turning of the head to the left side. The sensation ran down the left side of the neck to the arm and leg, and culminated in loss of consciousness and general convulsions. For a few days subsequent to this the patient suffered from headache, and felt generally unwell, but ultimately regained his former condition. For two and a half years, although maintaining his robust health, he was subject to daily recurrences of the paroxysmal twitchings of the left side of the face without loss of consciousness, and also to the more severe general convulsive seizures with loss of consciousness, which occurred on an average about once a month. Six months before admission spasmodic twitchings of the left hand and arm, without loss of consciousness, were observed and these have continued daily, alternating with the already mentioned twitchings of the face, the two, however, rarely occurring at the same time. Shortly afterwards weakness

of the left fingers, hand and forearm was experienced, which gradually increased to complete paralysis. Since the upper extremity began to be affected, there had been no recurrence of the general convulsive attacks with loss of consciousness. The patient was able to continue at work till August, 1884, when the weakness of the arm prevented him using his tools. Since then twitching of a like nature has taken place in the left leg, which usually supervenes upon, and is accompanied by, similar attacks in the arm on the same side. Quite recently the left lower extremity has been weak and the patient has walked a little lame.

*Present condition.*—On examination the patient was found in robust general health. His intelligence was unimpaired. All his organs and functions were normal except those about to be described. He suffered from frequent violent paroxysmal attacks of lancinating pain in the head, not localised but diffused over the vertex. There was nothing abnormal to be detected on the scalp or skull, and there was no special tenderness. On deep and hard pressure there was an area, not strictly defined, which seemed to be more sensitive than the neighbourhood. This was situated in the parietal region, close to the right of the sagittal suture, on a level with a line drawn vertically from the anterior portion of the external meatus of the ear. The movements of the eyeballs and pupils were normal; vision was normal, the patient being able to read No. 3 of Jaeger's types at twelve inches with the left, and No. 5 with the right eye. Examination of the fundi showed all the usual appearances of optic neuritis on both sides, most marked on the right, in the retina of which a number of small hæmorrhages were discernible. There was slight comparative immobility of the left side of the face, chiefly elicited by attempts at forced movements. The tongue when protruded pointed slightly to the left. Articulation was normal. The hearing was asserted by the patient to be normal, but was less acute in the right ear. A watch which on the left side was heard at three

feet, was only detected on the right at eight inches. The common sensibility of the head, and the other special senses were normal. There was complete paralysis of the left fingers, thumb and hand. The movements of the elbow-joint were very limited, and those of the shoulder impaired. There was no attempt at supination or pronation of the forearm. There was no trace of rigidity or wasting of the muscles. The irritability to mechanical stimulus of those of the forearm was markedly increased, and the temperature of the skin was lower on the left as compared with the right side. The left lower extremity was stated to be weaker than the right, but, when the patient lay in bed, its movements seemed much the same as those of the other, but were performed with more hesitation and less alacrity. When walking there was slight lameness, the toes were not completely cleared from the ground, so as to necessitate slight swinging of the leg in progression. The limbs were of equal size and the muscles of normal appearance. Their mechanical irritability and the knee-jerk phenomenon were greater on the left side, though somewhat excessive in both. The temperature of both legs was equal. The sensibility of the skin was everywhere normal, and the appearance of both sides of the body was the same.

*Progress of the case.*—While under observation in the hospital the condition described continued. The patient suffered frequently from paroxysmal attacks of lancinating pains in the head. These lasted sometimes for twelve or more hours at a time, and they were so violent that the patient was occasionally delirious and kept the whole ward disturbed with his cries. There were intervals during which he was entirely free from pain. He also suffered from seizures of very severe sickness not specially associated with the headaches. During these he vomited all food, and when the stomach was empty continued to retch with great violence. This would sometimes last for several days, causing great distress, and much reducing the strength of the patient. During residence in the hospital

the attacks of paroxysmal twitchings of the muscles were frequently observed. These occurred many times every day. The most common form was a rhythmical tremor which began in the first, second, and third fingers of the left hand, which afterwards spread to the thumb and wrist as far as the elbow. This continued for perhaps a minute, and then ceased, generally by the limb being held or rubbed. Another form began in the left angle of the mouth and side of the face, and a feeling as if the tongue was being contracted. These parts also continued to twitch for a minute or two. These two kinds of attacks rarely occurred at the same time, but took place independently of one another. Sometimes, but not commonly, the movements began in the face or arm, extended from the one to the other, and from thence down the side of the neck and body to the leg, so that the whole left side was convulsed without any loss of consciousness. The leg was never observed to be affected by itself.

*Diagnosis.*—The sequence of events described, with all the circumstances of the case, led to the diagnosis that there was an encephalic growth, probably of limited size, involving the cortex of the brain, and situated at the middle part of the fissure of Rolando.

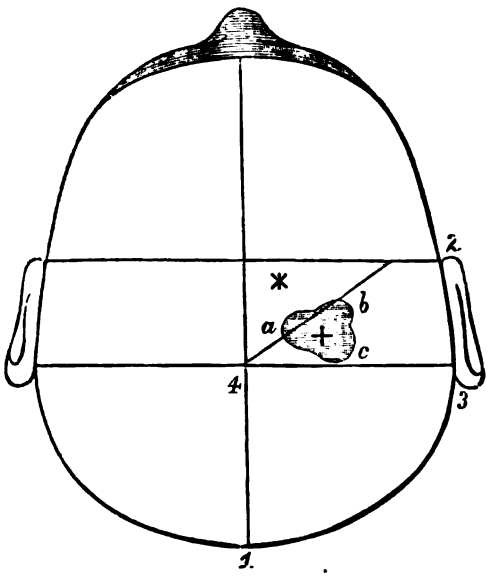
*Treatment.*—The patient was ordered the bromide and iodide of potassium, twenty grains of each, thrice daily, which he took for a month. Ice to the head gave no relief, and the vomiting was unrelieved by any treatment. The severe pain was ameliorated by hypodermic injections of morphia.

The terrible sufferings of the patient rendered life intolerable to him. All remedial measures having failed, and as it was obvious that his symptoms were extending, and that a fatal termination was not far distant, it was determined that an attempt be made to remove the morbid lesion. It was hoped that even if such a proceeding was not permanently successful it might alleviate some of the more pressing symptoms. The novelty and risks of the proposed treatment having been fully placed before the

patient and his friends, they readily consented to the adoption of any measures which offered any prospects of mitigating the urgent distress or of averting a certain death.

*Operation.*—In order to expose the cortex of the brain at the middle third of the fissure of Rolando the following procedures were adopted. A longitudinal line was drawn between the frontal and occipital protuberances, down the middle line of the scalp (Fig. 1, *1*). A second line was drawn at right angles to this at the level of the anterior border of the external meatus of the ear (Fig. 1, *2*). Parallel to this a third line was drawn at the level of the posterior

FIG. 1.




External surface of scalp. 1, 2, 3, 4. Lines to determine position of fissure of Rolando. + Theoretical and actual position of tumour. * Tender spot on scalp. *a*, *b*, *c*. Position and order of trephine openings.

border of the mastoid process, which reached the longitudinal line about two inches behind the second (Fig. 1, *3*). From the junction of the first and third lines, a fourth was

drawn diagonally downwards, reaching the second at a point two inches above the external meatus (Fig. 1, *4*). This diagonal line was believed to represent the direction of the fissure of Rolando. The spot where theoretically the centre of the trephine should have been placed was about half an inch behind the diagonal, and about one and a half inches from the longitudinal line (Fig. 1, +). As there was a tender point on the scalp about two inches anterior and to the inside of this (Fig. 1, *), it was determined to make the first opening in the skull between the two. (The order and position of the trephine openings are seen in Fig. 1, *a b c*.)

On November 25th, a trephine one inch in diameter was applied to this region (Fig. 1, *a*) and a circle of bone removed. The centre of the aperture was one and a quarter inches from the middle line and half an inch behind a line drawn vertically from the meatus of the ear. The dura mater was found normal in appearance. In this a crucial incision was made, through which the brain substance bulged, as was thought, abnormally. The surface appeared somewhat more yellow in colour than natural, but was otherwise apparently as in health. A second trephine opening was made, slightly overlapping the first (Fig. 1, *b*), external and slightly in front of it, and the angles thus left were rounded off with a chisel and hammer, the brain being protected by a copper spatula. The incision in the dura mater was prolonged, exposing an increased surface of brain but without further revelations. The trephine was applied a third time so as to join the two former openings posteriorly (Fig. 1, *c*), and when the edges were chipped off a triangular aperture with rounded angles was left, measuring two, by one and three quarter inches. The incision in the dura was then prolonged, exposing a surface of brain nearly the size of the opening in the skull, which presented the same appearance as that already described. Occupying most of this space and crossing it obliquely from above and behind, forwards and downwards, was a convolution, down the posterior aspect of which ran a large blood-vessel. Into the centre of, and parallel with

this convolution, an incision about three quarters of an inch in length was made with a scalpel. From an eighth to a quarter of an inch below the surface, a transparent lobulated solid tumour was seen, thinly incapsulated, but perfectly isolated from the surrounding brain substance. After prolonging the incision in the cortex, the surface and sides of the growth were easily separated by means of a narrow spatula of steel so tempered that it could be bent into any shape required. The mass was conical in shape the base being upwards. After its superficial portion was isolated, the finger was, as far as possible, inserted behind the tumour, and attempts made to enucleate it. In doing so the upper half broke across. A large Volkmann's sharp spoon was then employed to scrape out the deeper parts of the growth; and this was continued till all the morbid material was removed and apparently healthy brain matter only remained. This part of the operation was rendered difficult by the rapid welling of blood into the wound. No artery of any size spouted but there was a general oozing, which accumulated rapidly as soon as the sponge was removed. The cavity thus left was about one and a half inches in depth and of a size into which a pigeon's egg would fit. The hæmorrhage was arrested by applying over the cut surface a suitable electrode from an electro-cautery. The dura mater was then drawn together at its anterior part by a few carbolised silk sutures, and a drainage-tube of moderate calibre, made of india rubber, was inserted into the wound beneath the dura at its posterior border. The skin was brought accurately together, except where the tube lay, by silver wire and silk sutures. During the entire operation the carbolic spray was used, and both before and after, all the ordinary antiseptic precautions were taken.[1]

[1] The antiseptic precautions were as follows:—The patient's head having been previously shaved, except the very lowest part of the scalp quite below the occiput, the whole was thoroughly soaked with carbolic acid lotion (1—20), but particular attention was not directed to the sores left from some blisters at the upper part of the neck, which were not noticed till afterwards. After the patient was placed on the table the parts were again washed with 1—20 lotion, and the upper portion of the body was surrounded by carbolised

The wound was dressed with carbolic acid gauze, completely covering the scalp, and firmly fixed in position with bandages. During the entire operation, which lasted two hours, the patient took chloroform without a bad symptom, and no nervous phenomena were developed. Subsequent examination proved the tumour to be a glioma about the size of a walnut, presenting the usual microscopical appearances of that disease (Fig. 2).

FIG. 2.

Structure of glioma, from section by Dr. Hebb, about 400 diam.

*Progress of the case after operation.*—Half an hour after the operation the patient was in the usual drowsy condition which follows the administration of chloroform. He answered questions rationally and comprehended what was said to him. There was no increase of paralysis of the face or tongue, and articulation was as before. There was no trace of movement in any part of the left upper extremity, but the left leg could be moved at will, but to what extent it was not judged advisable to determine. Six hours after-

towels, one being also placed beneath the head. Instruments and hands were soaked in the same lotion and the spray was used throughout the operation.

wards the patient was quiet and comfortable. He had taken small quantities of milk and soda water, and had not been sick. Pulse 80, full and regular; temp. 98°. There was no pain in the head and only slight soreness of the wound was complained of. There was slight paresis of the left side of the face and occasional twitchings of both angles of the mouth, the cheeks, and the alæ of the nose. There had been no movements elsewhere.

November 26th (the day after the operation).—Pulse 56, of good strength and regular, morning temp. 98·4°. The patient had recovered from the effects of the anæsthetic, and was now quite sensible and intelligent. There had been occasional vomiting, shooting pains in the head, and twitchings on the left side of the face. During the night a fair amount of nourishment had been taken. The wound was dressed. A small amount of serum had soaked down to the edge of the dressing behind. The edges of the incision were in perfect apposition, and the scalp was quite flat.

27th.—The patient had passed a good night, and slept well. Had no return of vomiting; had occasional twitching of the left arm and side of the face, as well as occasional shooting pains in the head. The morning pulse 54, temp. 97·6°. Passed water, and the bowels acted freely after an enema. Took plenty of fluid nourishment. The patient was quiet, and at time of visit without pain. He was somewhat drowsy and apathetic, but perfectly intelligent, and answered questions with promptitude and accuracy. Articulation was normal. The movements of the eyeballs were natural. The pupils were somewhat dilated, equal, and contracted to light. There was distinct but slight paresis of the left side of the face. The tongue, when protruded, inclined somewhat to the left. The left upper extremity was throughout immovable. The left leg could be raised and pulled up when desired, but for fear of disturbing the patient, the amount of this was not fully tested. The left knee-jerk was markedly increased. The sensibility of the skin was everywhere

normal. There had been nowhere any twitchings for the last twenty-four hours. During the day there were occasional shooting pains in the head. The wound was left untouched as there was no appearance of discharge.

28th.—During last night the patient slept fairly well, but was occasionally disturbed by sharp pains in the head. Morning pulse 60, temp. 98·6°. The patient was bright and cheerful. He has had no twitchings of the face or limbs, but had suffered from occasional pains in the head. The paresis of the face was very slight, the articulation natural, and the condition of the limbs as before. He had taken plenty of nourishment. When the wound was dressed the discharge was found to consist of a small amount of thickish pink serum, with an obvious though faint smell. There was a distinct bulging of the scalp at the seat of operation, where it was slightly tender.

29th.—The patient had passed a good night. Morning temp. 98·6°, pulse 76. During the day there was no return of the pains in the head, or twitchings of the limbs. The general condition was as before. The patient was in good spirits, intelligent, and took food well. The left leg could be moved at will. During the evening there was some swelling of the eyelids and face, accompanied with smarting pain. On changing the dressings the lips of the wound was found swollen, and the discharge had a decidedly putrefactive smell. The lower lateral incision was opened up, and the drainage-tube removed and washed. From the openings some thick brown pus was squeezed. The scalp in the neighbourhood of the wound was somewhat œdematous. Dressings were reapplied as before.

30th.—The general condition was as before, except that to-day there were no movements of the left lower extremity. Morning temp. 99·4°, pulse 88. The swelling of the face and scalp was very considerable. In the morning the drainage-tube and several stitches were removed, and a dressing of wet boracic lint substituted for the gauze. The wound was freely syringed with carbolic

lotion (1—20) in which iodoform was suspended. The dressings were changed twice during the day, and on the last occasion all the stitches were removed. By this time there was hardly any trace of putrefactive smell. A hernia cerebri as large as half an orange, consisting of granular looking matter mixed with blood-clot, had protruded through the lips of the wound. Towards evening the swelling of the face and scalp had considerably diminished. At midnight the patient felt quite comfortable.

December 1st.—To-day an extended examination was made. Morning temp. 98·4°, pulse 88. The patient felt well in every respect. He was cheerful, perfectly intelligent, conversed freely, and took an interest in the details of his case. For some time there had been no trace of twitchings of the limbs or pains in the head. The swelling of the face and scalp had almost disappeared. The pupils were equal and normal. Vision was normal. The movement of the eyeballs was natural. The skin was cool and moist. The mouth and face were drawn slightly to the right on forced movement, and the tongue was protruded slightly to the left; articulation and hearing were as before. There was no movement whatever of any part of the left upper or lower extremities. The sensibility of the skin of the left limbs was considerably diminished to touch, but not lost. The left knee-jerk was still considerably exaggerated as compared to the right. The plantar reflex was the same on both sides. The general condition of the patient was excellent, and his appetite good. The urine was loaded with urates, but otherwise normal. The wound was dressed morning and evening. The hernia, which had somewhat increased in size, was freely treated with carbolic acid, iodoform, and a solution of chloride of zinc (gr. xl—℥j). The discharge had almost lost its offensive smell.

2nd.—The general condition as before. Now there was no trace of œdema of the face or scalp. Morning temp. 98·6°, pulse 88. The hernia cerebri appeared to be somewhat larger.

3rd.—The general condition as before. Morning temp. 97·6°, pulse 54. The greater portion of the hernia, which had now reached the size of half a cricket-ball, was clipped away with scissors. The parts removed consisted chiefly of granular matter and blood-clot, and apparently contained little true cerebral matter. They had a faint offensive smell. This removal was continued till a surface was reached where the tissues bled freely, which was only very slightly above the level of the scalp. This was freely treated with a strong solution of chloride of zinc and iodoform. The stump was dressed with boracic lint soaked in carbolic lotion, which was tucked under the edges of the flaps. The whole as at former dressings was enveloped in a mass of salicylic wool and firmly bandaged. Immediately after the dressing the temperature was 100°. In the evening when the wound was dressed again the discharge was found copious and watery.

4th.—The general condition as before. Morning temp. 98·4°, pulse 88. A cap of block tin was fitted over the hernia which bled slightly from small points all over its surface. The discharge was diminished and now quite odourless.

13th.—During the last ten days the general condition of the patient had continued to improve. On examination to-day he felt well in every respect. There had been no pain in the head or twitchings in the face or limbs. The appetite was excellent and all the organic functions were normally performed. The disposition was cheerful and the intelligence perfect. The patient conversed all day with the nurse, who could detect no signs of mental failure. There was still slight paresis of the left side of the face and tongue. The movements of the eyeballs were normal. The pupils were equal, and contracted normally to light and accommodation. Vision was apparently normal, and No. 7 of Jaeger's types could be read with the right and No. 4 with the left eye at twelve inches in rather a bad light. The optic neuritis still existed in both eyes, but was distinctly improved, the

hæmorrhages in the right having almost completely disappeared, and the swelling of the left disc being almost gone. The sensibility of the face was normal and all the special senses were as before the operation. The entire left upper extremity still remained completely paralysed, the muscles being limp and flaccid and without trace of rigidity. The left lower limb was also without voluntary movement. The sensibility of the skin of the entire left side, below the neck, was diminished to touch, but not altogether lost. The temperature of both sides was nearly the same. The mechanical irritability of the muscles and the tendon-reflexes on the left side were increased as compared to those on the right, and on the left there was well-marked ankle clonus. There was nowhere any rigidity or wasting of the muscles.

During the last ten days there was no essential change in the wound, except from day to day a gradual increase in size of the hernia. This now projected about an inch above the scalp. Its surface was smooth and clean, and the discharge was copious, colourless, and odourless. The margins of the mass were clipped away so that no mechanical obstruction might be offered to the contraction of the flaps, which process seemed to be in progress. In doing so a vascular part was soon reached, and a clear fluid in considerable quantity exuded. The deep surface of the flaps was covered with healthy granulations.

16th.—The patient continued well till last night, when he was restless, and felt his left arm and leg very cold. About six this morning he was seized with a rigor which lasted five minutes. Soon afterwards he suffered from shooting pains in the head and was very sick. Five hours after, the temperature was 100·2°, the pulse 100. The patient complained of severe pain in the frontal region. He was dull, apathetic, and nauseated. There were twitchings in the right arm and leg, and occasionally slight ones on both sides of the face. Until to-day there had been little change in the wound, except

that the flaps were gradually drawing together and tending to cut off the superficial part of the hernia. This morning it was found that the hernial mass had greatly increased in size. It was now rugged and of a dark colour. Attempts to remove portions of it were prevented by the profuse hæmorrhage. There was no putrefactive smell. In the evening it was found that the patient during the day had suffered much from pain in the head and vomiting. A hypodermic injection of morphia had been given in the afternoon, which had produced sleep for several hours. The temp. was 102·2°, the pulse 100. Later the temp. was 104·6°, the pulse 140.

17th.—Morning temp. 102·6°, pulse 140. Passed a very restless night, suffering much pain in the head, and in the right arm and leg. Was found pale and dull and very feverish. The patient understood what was said to him, but was slow to answer, and his replies were not easily understood. Another injection of morphia was given without inducing sleep or relieving pain. The hernia was found to have greatly increased in size, and was again about the size of half a cricket ball.

18th.—The patient passed a fairly quiet night. Morning temp. 101·2°, pulse 140. Has had little or no pain in the head, was quite intelligent and answered questions sensibly. There were no twitchings or increase of paralysis. He was feverish and very thirsty, but continued to take a good amount of fluid nourishment. The hernia was breaking down by a sloughing process, and a considerable quantity of semi-fluid detritus flowed from the cavities forming in the mass. This had again a slight putrefactive smell.

19th.—The general condition was the same as yesterday. The hernia was clipped off almost to a level with the bone, and a flat plate was placed and secured over the stump. In the evening the patient was very feverish and very talkative. He volubly related incidents in his past life, and carried on conversations quite sensibly with imaginary persons. He was very

restless and had no sleep. He was intelligent and answered questions correctly. Articulation was somewhat thick and indistinct. There was no apparent increase of paralysis, and the right limbs moved freely as before. He still continued to take plenty of nourishment. Evening temp. 103·6°, pulse 150.

20th.—Has had no sleep for twenty-four hours. Was still very restless and feverish, but had no pain. Was evidently weaker, but there were no new symptoms. Temp. 104°, pulse 150. When the eyelids were opened both eyes were seen deviating to the left, but could be voluntarily fixed in a straight line. No change in the wound.

21st.—The patient was evidently sinking. He was emaciated and a bedsore had developed on the right gluteal region. Still feverish, restless, and sleepless. He continued to talk volubly with a thick indistinct utterance. Temp. 104·4°, pulse 144, very weak. Was perfectly sensible. Bowels relaxed and motions passed involuntarily in bed. There was a general tremor of all the limbs, and the right side occasionally twitched.

22nd.—Had gradually become weaker. The breath had a sweatish smell and the skin a yellowish waxy appearance. The articulation was so indistinct as to be unintelligible. Had no pain, no sleep, was very feverish, and now refused food. Was still perfectly sensible.

23rd.—Since last report gradually sank, and died at 8 a.m. this morning. No new symptoms occurred and the patient was sensible to the end. The condition of the wound continued as last described.

*The post-mortem examination* (December 24th, thirty hours after death).—The body throughout was thin but not greatly emaciated. There was no special muscular wasting, the rigor mortis was well marked, and the skin was everywhere of a pale yellow colour and of waxy appearance. On the most prominent part of the left gluteal region was a circular patch three inches in diameter, of black discolouration. On cutting across and into this, it was

found to extend an inch in depth into the tissues, including a portion of muscle. On the right parietal region was an open wound of the scalp. This was of an irregular quadrilateral shape and measured three by two and a half inches. It reached to within half an inch of the middle line of the skull and its direction in its longest axis lay between this and the posterior margin of the ear. The edges were somewhat raised, everted, covered with healthy granulations, and for a quarter of an inch free, beyond which the skin was adherent to the bone. Elsewhere the scalp was healthy, and there were no signs of pus or putrefactive smell. The space between the edges of the wound was filled up by the base of the hernia which had been shaved off. This spread over the surface of the bone to which it was adherent, and had to be cut away with the knife. The removal of this exposed the aperture in the skull made by the trephine. This was of triangular shape with blunt rounded angles and measured two, by one and three quarter inches. Its longer axis lay almost exactly between the parietal protuberance and the central line of the skull, reaching to within about half an inch of both. The edge of the bone was perfectly healthy and presented the appearance of a clean cut. The skull cap was removed in the usual manner and the brain and cord were taken out, when it was seen that the inner aspect of the arachnoid at the base of the cranium was lined by a layer of pale yellow, coagulated, recent lymph. This was most abundant in the right middle fossa and over the base of the sphenoid bone. It also extended for a short way down the spinal canal. The dura mater of the base was somewhat thickened but otherwise normal.

*The brain.*—On inspecting the base of the brain a thin layer of lymph was found spread over the surface of the arachnoid. This was most abundant over the base of the right temporo-sphenoidal lobe, over the pons and medulla, and down the upper part of the spinal cord for about an inch. It reached forwards, but less in amount, to the bases of the frontal lobes, sideways to the inner

edge of the left temporo-sphenoidal lobe, and backwards as far as the anterior border of the cerebellum. The outer edges of the cerebellum, most of the left temporo-sphenoidal lobe, and the under surface of the frontal lobes were entirely free from the effusion. This deposit of lymph extended from the base of the brain over the right temporo-sphenoidal lobe, and could then be followed by a tract about an inch wide to the under surface of the wound, from which it evidently emanated. The membranes covering the under surface of the right temporo-sphenoidal lobe were markedly congested and their vessels dilated. There was also slight injection of those at the under surface of the cerebellum. Elsewhere the membranes of the base were healthy. The convolutions of the base of the brain were everywhere normal in appearance, except that those of the right temporo-sphenoidal lobe were somewhat more flat and the sulci less deep than those of the opposite side. The consistency of the cortex was here everywhere intact, except a patch of slight softening about the size of a sixpenny piece at the most anterior extremity of the temporo-sphenoidal lobe. The external surface of the dura mater covering the hemispheres was on both sides normal in appearance, except over the right parietal region, where the wound had been made, through which protruded the base of the hernia cerebri. This corresponded almost exactly in size, shape, and position with the aperture in the skull and measured two and a half by two inches. The free edge of the dura was adherent all round to the cut surface of the bone. On reflecting this from the brain it was found normal in every respect on the left side. On the right it was somewhat thickened throughout, and very considerably so immediately round the wound, especially behind and below. Here also in several places there were recent adhesions of the two layers of the arachnoid which were readily torn across without force. Traces of lymph were found scattered over the whole hemisphere, but chiefly over the occipital and temporo-sphenoidal lobes. It was most abundant in

a narrow tract stretching from the lower border of the wound, proceeding downwards to the base of the brain as already described. Above and in front of the wound there was no appearance of inflammatory exudation. Throughout the left side there was slight subarachnoid effusion. The anterior lobes were pale and normal in colour. Behind a vertical line drawn through the bases of the frontal convolutions the membranes at the upper part of the cerebrum were of a pinker colour, and their vessels more injected with blood, than in front and below; otherwise their appearance was normal. The convolutions on this side appeared to be slightly flattened and the sulci somewhat shallow, otherwise they were normal. On the right side the membranes above and in front of the wound were precisely the same as on the left. Behind and below, and especially over the superior part of the temporo-sphenoidal convolutions, they were intensely congested. The convolutions of the frontal and parietal regions on this side, both in appearance and consistency, were in all respects the same as those on the other, and they were equally voluminous on both sides, but the parietal area had a shrunken appearance as if it had fallen inwards. In the centre of this, and occupying the position of the fissure of Rolando, was the wound in the brain. It corresponded in position to the hole in the skull, but was a trifle larger, measuring two and a half by two inches, the longest axis being directed somewhat obliquely from above downwards.

The destruction of the cerebral cortex is illustrated in the accompanying diagrams (Figs. 3 and 4), and will be seen to involve, first, the entire length and thickness of the ascending parietal convolution with the exception of a small portion of its superior and inferior extremities, both of which remained intact; secondly, almost the entire upper third of the ascending frontal convolution, and the posterior portion of its upper half; and thirdly, the anterior third of the gyrus supramarginalis. This deficiency in the grey matter was occupied by the rough material

constituting the stump of the hernia cerebri which projected about half an inch beyond the surface of the brain. Surrounding and closely adjacent to this on its anterior, superior, and posterior aspects, the cerebral cortex was normal in appearance and firm in consistency. The margin of the aperture in the grey matter was sharply cut, slightly folded inwards, and its inner edges were adherent to the hernia. At the inferior border of the wound the convolutions, although normal in appearance, were

FIG. 8.

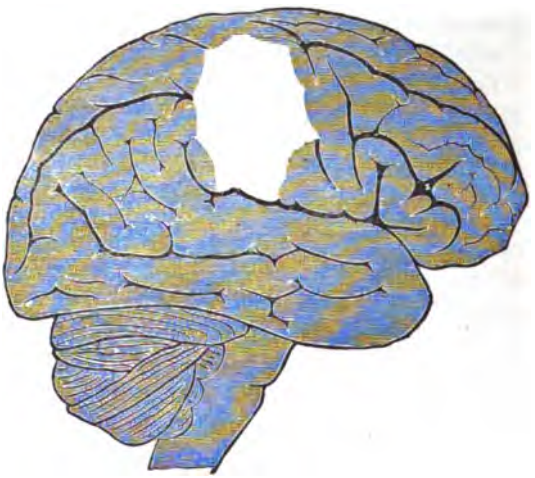


Diagram showing position and extent of cerebral cortex destroyed as seen from without. The white space occupied by stump of hernia cerebri.

slightly softened to the touch, and this softening extended so as to include the superior temporo-sphenoidal convolution, below which the cortex was of firm and normal consistence. On the inner aspect of the right hemisphere there was a circular depression, about the size of a sixpenny piece, without softening, and which appeared as if the part had fallen inwards. This involved the terminal portion of the fissure of Rolando, and a part of the sulcus caloso-marginalis, a small portion of the

gyrus fornicatus, the posterior part of the marginal convolution, and the anterior border of the quadrilateral lobule. A transverse section was made across the brain through the ascending parietal convolution in the direction of the fissure of Rolando. The deficiency in the cortex is seen in Fig. 4 to consist of complete absence of that part which corresponds to the middle parietal fasciculus with the inferior portion of the superior, and the superior portion

FIG. 4.

Diagram of transverse section through ascending parietal convolution showing destruction of cerebral cortex and corona radiata. The white space occupied by granular matter of hernia cerebri.

of the inferior parietal fasciculi. The spaces thus left had partially collapsed, and were filled with new formation from which the hernia cerebri sprang. This morbid condition had in an irregular and unequal manner spread inwards, reaching as far as the upper margins of the corpus callosum and internal capsule. The brain having undergone the process of hardening it was not easy to exactly limit the softening, which seemed to be confined entirely to the centrum ovale. The lower portion of the

inferior, and the inner portion of the superior parietal fasciculi were uninjured. So also were the insular lobe, the internal and external capsules, the corpus callosum, the optic thalamus, and the lenticular and caudate nuclei. Whether the lateral ventricle had been opened into it is difficult to say. There was no collection of foreign matter in its interior; at the same time the morbid process had extended in close proximity to it.

A histological examination of the different tissues was made by Dr. Hebb, who reported their condition to be as follows:

"The appearance of a section of the glioma has already been referred to (Fig. 2). The hernia cerebri and subjacent tissues consisted of blood-clot, granular matter, and disorganised nervous tissue. The cortex in the neighbourhood of the wound presented the appearance of inflammatory change in its pia mater and superficial layer, but was otherwise normal. Elsewhere the structure of the brain was healthy, and nowhere was there any evidence of gliomatous disease. The retinæ and optic nerves presented all the usual appearances of neuro-retinitis in a well marked but not advanced stage. The membranes of the cord had undergone marked change. The inter-meningeal space at the upper cervical region was filled with pus. In the cervical and dorsal regions the membrane was considerably thickened, which condition gradually diminished from above downwards. In the grey matter of the cord there was evidence of nuclear activity, apparently of quite recent origin, otherwise it was healthy. There was no trace of descending sclerosis."

*Commentary.*—In commenting on the preceding series of facts it will be convenient to discuss the points of interest under the following heads: (1) Diagnosis, (2) the surgical treatment, (3) the clinical phenomena after the operation, (4) revelations of the autopsy, physiologically and pathologically considered.

1. *Diagnosis.*—All the circumstances of this case pointed to an encephalic growth on the right side. This was

more especially evidenced by the slow and insidious invasion of the symptoms, the gradual progress and special distribution of the paralysis, the violent intracranial pain, the uncontrollable attacks of vomiting, and the double optic neuritis. Whether a tumour on the right side of the brain was caused by the blow on the left side of the head a year previous to the development of the first signs of ill-health, must remain uncertain.

It was also concluded that the morbid lesion involved the cortical substance, because certain motor phenomena were developed, and certain motor districts implicated after a definite method and in definite order; the paralysis was unaccompanied by any alterations in sensibility; and above all, because of the existence of certain paroxysmal seizures of local convulsion, without loss of consciousness, which were eminently suggestive of irritation of cerebral grey matter.

The special seat of the lesion was further believed to be the middle part of the right fissure of Rolando. This conclusion was arrived at by the consideration of the combined revelations of physiological experiment and clinical observation. After centuries of doubt and confusion on the subject of cerebral localisation, quite recent investigations have at last rendered it certain that around this sulcus are grouped those nervous areas which preside over the movements of the other side of the body. Adopting the topography of the brain constructed by Professor Ferrier as the result of his well-known researches, it may be said in general terms, that the motor centres which govern the voluntary movements of the lips and tongue are situated in the lower portions of the ascending parietal and frontal convolutions. Higher up in the same gyri are the areas for the muscles of the face. Occupying the middle portion and nearly the whole extent of the ascending parietal convolution are the centres of the fingers and hand. In the middle of the ascending frontal convolution are those elements which originate movements of the arm and upper arm, including flexion, pronation, and supina-

tion of the forearm. At the superior and posterior aspect of the ascending parietal convolution is the centre for the lower extremity, and at the upper and anterior portion of the ascending frontal convolution are centres for complex movements of both the upper and lower limbs. Now, in the case before us there was complete paralysis of the fingers and hand, with inability to pronate and supinate the forearm, there was partial paresis of the movements of the elbow, and weakness of those of the shoulder-joint. There was also slight paresis of the leg and one side of the face. Accompanying all these there were paroxysmal convulsions in all these regions, occurring either singly or in definite order one after the other. These phenomena were to be accounted for by an extensive but not absolutely complete destruction of the motor centres of the fingers, hand, and forearm, with slight encroachment on, and irritation of, those of the face, upper arm, and leg. A very definite localisation of the disease was thus permitted, and the tumour was pronounced to have occupied the whole thickness of the middle two fourths of the ascending parietal convolution, and a portion of the adjoining upper half of the ascending frontal convolution. The morbid lesion, whilst almost completely destroying these areas, in addition modified the functions and caused irritation of those in their neighbourhood, without seriously involving their structure, namely, the superior and inferior extremities of the ascending parietal and frontal convolutions, and the postero-parietal lobule.

Having thus accurately localised the position of the tumour its size could as a consequence be approximated. Assuming the disease to be limited to the cortex at the point already indicated, the fact that the centres of the leg above, of the face and tongue below, of the sense of sight behind, and of the movements of the eyeballs in front, were not seriously involved, proved that the growth was of limited size. A glance at the relative position and size of the convolutions of the human cerebrum indicate that a foreign body occupying such a position could not, roughly speaking,

exceed some two inches in diameter. It was probable that the growth took its origin in the lower third of the ascending parietal convolution, and as it increased in size spread upwards and backwards, further involving its substance and part of the ascending frontal convolution, finally reaching the lower edge of the postero-parietal lobule. Such disease might therefore be represented by an ovoid mass the size and shape of a pigeon's egg, lying obliquely in the fissure of Rolando. This theoretical reasoning arrived at before the operation, subsequently proved to have been substantially correct.

The question finally arose whether the tumour was confined to the cortex or whether it was situated in the centrum ovale below, and from thence invaded the grey matter. It was not forgotten that a slowly-growing mass, reaching considerable dimensions, might develop in the conducting elements of the brain, without causing symptoms capable of exact definition. It was also fully recognised that a small tumour immediately under the cortex and involving its under surface might cause precisely the same symptoms as one limited to the grey matter. From an operative point of view the existence of even a large growth, which in this case was improbable, would not necessarily interfere with the procedure, because in that case little harm could be done to the life of the patient, and his urgent symptoms, on the other hand, might be relieved by the simple process of trephining. If the tumour was small the grey matter could be incised and the mass removed. As a matter of fact this last is what actually was done in the case under consideration, the growth being found in the centrum ovale, under the cortex, involving the convolutions before determined.

As to the probable nature of the tumour, the age of the patient, the absence of syphilis, and the slow growth of the disease suggested glioma, but on this point no definite conclusions were hazarded.

2. *The surgical treatment.*—For the purpose of removing the tumour from the brain, three trephine holes

were made in the skull, because after the first piece of bone was taken away and no superficial disease discovered, it was thought advisable to enlarge the opening, to further expose the brain and to make room for completing the operation. The aperture made by the three removals, even when the angles were chipped off, only measured $2 \times 1\frac{3}{4}$ inches. The tumour was found exactly in the centre of the opening, and in the position corresponding with theoretical calculation and measurement. It had no relation whatever to the tender spot on the skull, and, had attention been confined exclusively to that, the result would have been entirely misleading. The process of trephining involved no difficulties, but it exemplified the advantages of the chisel and hammer over Hay's saw for rounding off corners of bone. In similar operations it might be advisable in the future to employ a larger trephine. One convolution only being exposed during the operation, there was at the time some question as to whether it was the ascending frontal or parietal. This doubt arose from the circumstance that in the attempt to approach the tender spot, the theoretical position had been slightly departed from, and the trephine advanced and raised, so as to come between the two. Accordingly for the moment this convolution was thought to be the ascending frontal, the aperture in the bone being so small that its relations could not be seen. After death, however, it was apparent that the convolution which had been incised was that in which from the first the disease had been diagnosed to exist, namely, the ascending parietal. There was no external appearance of disease about this part of the cortex except that it seemed swollen, less glossy, and less vascular than natural. An incision into this showed the morbid growth to be immediately under the surface, and almost completely involving the entire thickness of the cortex. On clearing the superficial parts of the growth, a small spatula, neither sharp nor blunt, and so tempered that it would keep any shape given it, was found a most serviceable instrument. Such is preferable to the

use of the cautery, as the latter so chars the parts as to modify their natural appearance, and thus prevent a differentiation between healthy and diseased tissues. It may be questioned whether it was advisable to arrest the hæmorrhage from the interior of the wound by means of the galvano-cautery. Such a proceeding leads of necessity to the formation of a considerable amount of detritus which may afterwards prove detrimental. The bleeding moreover was not severe and would no doubt have become arrested by natural means. The advisability of introducing a drainage-tube may also be questioned. It was not judged safe to completely close so large a wound, distended as it must have been with accumulated serum and blood. Moreover, had putrefaction not occurred it is not likely that the soft india-rubber tube would have caused any serious irritation. The argument in favour of complete closure of the wound, so strongly advocated by those whose experience is confined to operations on the brains of monkeys, is, it is maintained, not convincing when applied to a large injury in the human subject, the more rapid healing of the tissues of the lower animals being a matter of common knowledge. Another point of doubt is the propriety of introducing sutures into the dura mater. The most important matter for discussion, however, is the occurrence of putrefaction, which undoubtedly appeared in the wound some days after the operation. This, it may be maintained, was the cause of the inflammation and consequent hernia cerebri. All the usual antiseptic precautions were taken during the operation, and the only flaws in its strict application which suggest themselves are, first, that the scalp was not sufficiently purified prior to the operation, and second, that no special measures were taken to carbolise the galvano-cautery apparatus. In future operations of this nature it is strongly urged that surgeons should not only employ carbolic acid, but also a solution of corrosive sublimate as antiseptics, and that the scalp should not only be rubbed with these, but soaked with them for some hours previously.

There may have been other sources of septic contagion in the washing of the sponges, or from the blisters on the neck which escaped observation. It may be doubted whether the putrefaction was ever completely subdued; the fact of the meningitis occurring at last, and that of a smell having again become apparent after the attempt at removal of the second protrusion, point probably to a continued septic infection. As to the hernia two observations only have to be made. First, it was remarkable that the discharge continued for such a long time to be so copious and so watery, which suggested the idea of its being cerebro-spinal fluid. Secondly, there was a difficulty in shaving it off owing to the enormous size of its base, and to the danger of serious hæmorrhage.

3. *Clinical phenomena following the operation.*—The patient, on recovery from the effects of chloroform after the operation, was found perfectly intelligent, the former pain in the head, and violent twitchings in the limbs, had disappeared and never returned, there was no increase of the paralysis of the face or leg, and all the organic functions remained normal. The only change which had taken place was completion of the paresis of the upper extremity, which was now paralysed throughout. This was evidently due to the unavoidable destruction of the remaining arm centres in the removal of the tumour. Otherwise the neighbouring brain matter had not been injured, as was evidenced by all other functions remaining intact. The surgical operation itself in no way injured the nervous centres with the exceptions mentioned, while it immediately relieved all the distressing symptoms. This satisfactory condition remained unchanged for four days, when the discharge from the wound was found to have a putrid smell. Coincident with this began the hernia cerebri, and following its development, arose fresh symptoms in the shape of paresis of the left leg and partial anæsthesia of one half of the body. These were probably due to the effects of simple pressure, and possibly to the subsequent secondary softening of the conducting

fibres caused by it. That the inflammatory condition which led to this was purely local was shown by the fact that, with the above exception, the condition of the patient remained in all respects as before the operation. The temperature never reached 100° or the pulse 100 beats per minute. The intelligence was absolutely intact and the appetite and general condition in every respect satisfactory. The patient had lost all pains in his head, all traces of twitchings of the limbs, and all his severe attacks of vomiting. Even the double optic neuritis had markedly diminished. This state continued daily to improve till the twenty-first day, when suddenly the patient was seized with a rigor followed by fever and all the symptoms of meningitis from which he died a week afterwards. This inflammation was afterwards seen to be local and due to septic matter from the wound causing irritation of certain areas of the cerebral membranes. If putrefaction was the sole cause of this condition, hope may be entertained that by its prevention in other cases a more satisfactory termination may be looked for. Although meningitis continued to a fatal end, no new nervous symptoms supervened, the absence of which was probably due to the presence of a hole in the skull, through which excess of pressure was relieved.

4. *Revelations of the autopsy.*—After death, inspection of the parts showed that the brain was practically everywhere healthy except the area injured by the operation and the membranes in its immediate neighbourhood. From its lower border a narrow tract of recently effused lymph extended downwards by the temporo-sphenoidal lobe towards the base of the skull, over a large portion of which it spread, leaving the adjacent parts healthy. It was therefore obvious that this condition was produced by irritating matter from the interior of the wound, flowing downwards between the layers of the arachnoid, accumulating at the base, and by its presence causing meningitis in its track. The local inflammation of the wound had so opened out the parts and separated the adhesions as to permit the

discharge to percolate into the cranial cavity, but not till three weeks after the operation. Had this not occurred there is no reason why the healing process should not have been maintained, and the entire wound become ultimately cicatrised. The patient would then have continued permanently in a satisfactory condition, and escaped the secondary and fatal complication. The recovery from serious surgical injuries to the brain-substance of man, as well as experimental researches on that of animals, show that such a termination is perfectly possible.

The cortical substance at the edges of the wound in the brain was firm and healthy, except at the inferior border, which was slightly softened, probably from infiltration of the meningeal effusion. The deficiency in the grey matter was clearly defined and the portions of absent convolutions could be accurately limited. On the subject of central localisation only general conclusions can be drawn, as the destruction was not limited to the cortex, but in great part was situated in the centrum ovale below. The fibres, however, thus injured were those corresponding to the grey matter above, and may therefore be said to represent the conducting media of the higher centres. The symptoms immediately before the death of the patient, as far as they go, entirely harmonise with those which have already been determined by experimental inquiry to arise from corresponding lesions of cortical matter, with others superadded, which can be easily explained by the processes of pressure and softening in the neighbourhood. The inferior extremities of the ascending frontal and parietal convolutions being found only very slightly involved, accounts for the almost total absence of orolingual symptoms during life. The almost total destruction of the remainder of the ascending parietal convolution explains the complete paralysis of the fingers and hand, and the partial paresis of the face. The lesion of the middle third of the ascending frontal convolution produced the immobility of the elbow- and shoulder-joints, and the loss of pronation and supination in the forearm. The

almost complete immunity from disease of the lower part of this gyrus permitted the nearly natural movements of the face, lips, and tongue during life. The bases of the three frontal convolutions were perfectly healthy, but a day or two before death temporary conjugate deviation of the eyeballs was observed, both being turned towards the left, which was probably due to irritation of these regions by the neighbouring disease. At no time was there any paralysis of the muscles of the eyeballs. The postero-parietal lobule was found almost intact, its anterior margin only being involved in the wound. For some days after the operation the patient moved his left leg freely, and it was only after the appearance of the hernia that the limb became paralysed. This was therefore due not to destruction of the cortical centre of the lower extremity, but to pressure and softening within the wound. This was evidenced by the sinking in of the healthy convolutions on the inner aspect of the hemisphere at a point exactly corresponding with the situation of the conducting fibres of this region. The anterior portion of the supra-marginal gyrus was absent. This convolution Professor Ferrier associates with the sense of sight. In this case there was no evidence of any serious impairment of vision or hemiopia, although the patient saw better with the left than with the right eye. There was, however, double optic neuritis, most marked on the right side. The deficiency in sight was evidently due to this and not to a central lesion, in which case the weakness of vision would have been chiefly in the opposite or left eye. It is therefore probable that no appreciable loss of function could be attributed to the disorganisation of a portion of the right supra-marginal gyrus. It is, however, to be observed that the convolution was only partially destroyed, and Professor Ferrier has shown that even when it is completely obliterated on one side the consequent blindness on the other is only temporary, the opposite centre apparently rapidly compensating for the loss. Shortly before death the patient, though sensible, talked very volubly, carried on

conversation with imaginary persons, and recited the most elaborate and yet perfectly coherent adventures. May these not have been the result of visual hallucinations, and due to irritation of this centre ?[1]

Although the right superior temporo-sphenoidal convolution was somewhat softened it was not so to any great extent, and it was probably recent and due to mechanical infiltration. During life the hearing of the left ear was perfect. The comparative deafness on the right side was due to deficiency in the auditory apparatus and not to a central lesion.

The destruction of the centrum ovale for the main part corresponded with that of the cortical substance above. Its exact limits were difficult to define owing to the gradual softening in the neighbourhood. The internal capsule, corpus callosum and basal ganglia were, however, intact. So also was the remainder of the brain. The intellect, other senses, with all the organs and functions of the body except those already detailed, remained normal till the last.

Such are the main points of interest and reflections concerning a case which throughout has been a source of great anxiety and responsibility. This has chiefly been due to the fact that we have not had the advantage of any precedent of a like nature to guide us in our methods of procedure. Operations on the brain-substance have not been uncommon in the history of medicine, but these have hitherto been performed either for the relief of surgical injuries, or for disease indicated by local manifestations. We have nowhere been able to discover the recorded example of a case where a cerebral tumour was diagnosed by the symptoms observed, without visible or tangible external signs, and was in consequence operated on and successfully removed. Since this has been accomplished in the present instance, the public papers have asserted that the same has already been carried out on

[1] In connection with the sense of sight, the fact may be noted that the optic neuritis was most severe on the side of the cerebral lesion.

several occasions in the Royal Infirmary of Glasgow. To this it can only be said that up to the present date no report of such proceedings is to be found in medical or scientific literature.

In conclusion, we would observe that, although unfortunately in this instance life was not permanently preserved, the experience we have gained by this case leads us to the belief that there is an encouraging prospect for the future of cerebral medicine and surgery, and that as a tumour of the brain can be diagnosed with precision and successfully removed without immediate danger to life, we confidently anticipate that under more favorable circumstances the operation will be performed with lasting benefit to the patient.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 438.)

# THE

# EXPERIMENTAL PRODUCTION OF CHOREA

AND OTHER RESULTS OF

# CAPILLARY EMBOLISM OF THE BRAIN AND CORD.[1]

BY

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In December, 1883, at the completion of my Medical Registrarship at the Hospital for Sick Children I resolved to undertake the investigation of which the present communication is the first record. It seemed to me that the possible effects of genuine capillary embolism in cerebro-spinal pathology had been far from adequately considered. I commenced my experimental investigation on the 1st of October, 1884, at the Brown Institution, to which my friend Mr. Victor Horsley had just been appointed Professor Superintendent.

The material with which I began to work was guinea-pigs and rabbits as subjects and bismuth and starch as

[1] Towards the expenses of this research a grant was made by the British Medical Association on the recommendation of the Scientific Grants Committee of the Association.

artificial emboli. My object in choosing bismuth and starch was the circumstance that each could be recognised in the nervous tissues by means of a simple chemical reaction. Ammonium sulphide would turn the crystals of subnitrate of bismuth black, whilst iodine would impart a blue-black colour to the granules of which starch is composed.

I found it necessary to make a microscopical examination of many particulate substances having the following qualities. First of all the powder must not be too heavy or the temporary suspension in a "neutral" salt solution would be impossible. Secondly, the particles must be capable of easy recognition, or hours of unnecessary labour would be required to discover their locality after injection; and lastly, the size of the particles must not be too great, or vessels larger than capillaries would be blocked. Another consideration was that particles must be chosen which had no great tendency to agglomerate when the powder was distributed through a ·75 per cent. salt solution. I may say at once that the particles with which I have obtained the best results have been the granules of potato starch, those of arrowroot, and those of carmine. The individual granules of the last-named powder are very much smaller than a red blood-corpuscle, but a suspension of the powder in salt solution always contained many granular masses larger than the average red blood-corpuscle.

The next difficulty which had to be overcome was the method of production of embolism. It was obvious that direct entry of the arterial cerebral and spinal circulation was most necessary, or at all events most advisable. After many dissections and much deliberation I came to the conclusion that in rabbits and guinea-pigs the only way of obtaining results without having recourse to too large an operation was to inject into the aorta by way of the common carotid arteries. I found that by slowly injecting the particulate liquid into one or other common carotid the aortic flood nearly always carried the minute

particles up one or both vertebral arteries, which are given off in rabbits and guinea-pigs much in the same way as they are in the human subject. The internal carotid is a very slender branch of the common carotid, and injection upwards from the common carotid always failed, in the animals mentioned, to get into the brain or circle of Willis.

The operation which I performed on rabbits and guinea-pigs, cats, and dogs was as follows: The animal having been first anæsthetised by means of ether or chloroform, it was fixed upon the operation table and an incision made a little to one side of the median line of the neck, say the right. The skin and fascia were then divided with the knife and scissors and the muscles separated by blind hooks. The carotid artery was then explored and cleaned and laid upon a platform of white paper. A ligature was next put on the vessel at the upper end of the wound, and a clip served to close the lumen of the artery at the lower end of the wound. By means of a pair of scissors a snip was then made in the wall of the artery nearer the upper than the lower end of the exposed vessel. The next point was the introduction and tying in of a fine steel blunt-pointed cannula adapted from an ordinary hypodermic syringe. The cannula having been introduced the body of the syringe was filled with the particulate liquid, then the clip was released from the artery and a drop of blood was allowed to flow out at the free end of the cannula; the syringe was then promptly fitted on to the cannula and the injection slowly made. The blood pressure is so low that it can easily be overcome without using any force to the piston of the syringe. The injection completed, a moment or two is spent in waiting till the flush of blood had washed all the injected fluid out of the proximal end of the carotid artery, when the clip is reapplied, the cannula removed, the vessel ligatured, and the wound closed and sutured. The surface of the wound was washed with a solution of carbolic acid, and every care was taken to prevent septic inoculation, though the spray was not generally used.

I shall divide the information which I have to communicate into three parts. The first division will treat of the clinical phenomena which are apparently dependent on capillary embolism of the brain and spinal cord. The second part will deal with the anatomical changes caused by this process. The third section will be concerned with remarks bearing on the pathology of the clinical and anatomical facts, and more especially of those facts as they relate to chorea. This division of matter will not, however, be strictly adhered to.

## *Clinical phenomena.*

1. The most important observation which has come out as the result of these experiments is the production of involuntary movements indistinguishable from those of human chorea, allowance being made for differences of anatomy. The choreic movements observed in the animals experimented on by me were, I believe, not due to capillary embolism of the brain proper. Whenever they were present emboli were always found in the capillaries of the spinal cord.

2. Some form of "uncontrollable" movement was observed in almost every experiment in which the capillary emboli lodged in the brain. Sometimes the animal rolled round the longitudinal axis of the body. In other animals the movement was round an imaginary circle with the centre to one side of the animal; the movement in these cases was always in one and the same direction. Other animals made somersaults, revolving round a transverse axis of their trunks. Other animals, again, had a kind of "rocket" movement in which they darted forwards, generally in a direction obliquely with the long axis of their bodies. These "forced" movements were exactly like those due to section of various parts of the brain and which have long been known to physiologists. That capillary embolism can give rise to such movements of

rotation is, so far as I have been able to ascertain, a new fact, though there appears to me to be nothing strange in the occurrence of such movements as the result of capillary embolism. I have observed these "forced" movements in the guinea-pig, cat, and dog. These movements were unattended by any appreciable paralysis or tonic spasm. Not infrequently there was an abnormal carriage of the head and it seemed as though the cephalic posture afforded some kind of comfort to the animal, which appeared to be very giddy when the head was placed in its normal position, which could easily be done. As a rule this abnormal carriage was due to a rotation of the head round the long axis of the neck, so that one side of the face was directed towards the floor whilst the nose was projected forwards and upwards.

3. In the majority of cases the animals experimented on by me have had marked nystagmus, either horizontal or oblique; this has been observed in the four kinds of animals used. The rhythmical movements of the eyes were not absolutely constant, and generally tended to disappear when the animals lived more than a day.[1] The rate varied somewhat; a common rate was about eight to-and-fro small vibrations in five seconds. So far as my notes go the affection was always bilateral. (Well-marked conjugate deviation of the eyes was observed in a dog.)

4. Twitchings of irregularly distributed groups of muscles were observed in most of the animals experimented on. These twitches were observed in the face, neck, head, upper part of trunk and forelegs. A few of the artificial emboli were found in sections of the upper part of the spinal cord in these cases.

*Amplification of* 1 *and* 4.—As these twitches in my first experiments were not a marked feature I did not attach much importance to them as throwing light on the pathology of chorea. The main point to prove in connection with chorea as due to embolism is the fact that a succession of clonic spasms can be caused by the embolism

[1] *Vide* Adamkiewicz, Zur Lehre Gehirndruck.

because a series of movements of different groups of muscles is the most essential feature of chorea.

I shall now describe more in detail the case of a cat which, I believe, settles the point in question—that chorea may be due to capillary embolism.

A black female cat was submitted to experiment at 11.30 a.m. on February 18th, 1885. The operation was finished and the animal came to at 12 noon. As soon as consciousness was fully restored the choreoid movements were observed. The right foreleg was chiefly affected, but the movements were present in the face, head, and neck, upper half of trunk, shoulders, and forelegs. There was also horizontal nystagmus. The muscular contractions were thus arranged: the face would twitch into a grimace in which the angle of the mouth would be drawn outwards and upwards; the head would be jerked in one direction and then the shoulders would be shrugged and the arm drawn inwards and paw contract and the trunk jerked all in succession. The movements were disorderly. The movements of this cat were such that to my observation and many others who observed them they could not be distinguished from a typical case of chorea in the human being. Naturally the characteristic pronations and supinations of the forearm were not present for the very obvious reason that cats do not possess such movements. But there were other symptoms present. The reflex action in the upper limbs was greatly exaggerated, whilst the reflex action of the lower limbs was simply normal. A tap on the lower limb caused strong movement of the upper whilst giving rise to a normal reflex movement in its own muscles.

The animal seemed to be in a drowsy, giddy state, and preferred to lie on its left side. When disturbed it always sprang round in a circle to the left with the base of the tail as a fixed centre. Moreover, when sat upon its haunches it moved with good force and apparently viciously all its limbs. Careful observation during the whole eight hours it lived failed to detect any sign of actual paralysis

or tonic spasm. No loss of sensation could be made out in any part. The pupils were natural and acted apparently naturally. The temperature was 96° in the vagina, and observations in the axillæ did not detect any appreciable difference on the two sides of the body. The respirations were 30, the pulse 120. The movements continued, increasing in frequency, till death, which was no doubt due to collapse. At the autopsy no naked-eye lesion could be detected; the right common carotid was the artery used, and the injection was made downwards towards the heart, and the emboli (arrowroot granules) must have gone chiefly up the vertebral arteries. Emboli were found in all parts of the brain. There were emboli in the upper half of the spinal cord; they were not numerous, generally speaking two or three granules were found in almost every section of the cervical enlargement of the cord. None were detected in the lumbar region. (Specimens shown of the emboli in this case.) It is important, I think, to associate the presence of emboli in the cervical part of the spinal cord with the greater affection of the muscles supplied from this part and with the increased reflex excitability. There were no choreoid movements in the lower limbs, no exaggeration of reflexes, and no emboli in the spinal cord of this region. Can we resist the conclusion that the clinical symptoms are related etiologically to the presence of the emboli in the spinal cord? I think not.[1]

5. In several of the animals experimented on the head and neck was the seat of almost rhythmical to-and-fro movements which lasted till death or until the animal was killed. The longest duration was five days in a guinea-pig. In one guinea-pig where carmine was injected the

[1] I have notes of at least two cases of typical severe chorea in girls in which a tap on the upper part of the back of the forearm produced movement of the index-finger, or middle, or ring- and little fingers, according to the site percussed; in these two cases the knee-jerks were exaggerated so that their equivalents could be obtained by drawing down the patella with the forefinger and percussing the straining forefinger.

rotation movements persisted for thirty-six hours, and then completely passed away. The animal was allowed to live for ten days; it was then killed. Carmine was found blocking many of the capillaries of the pons and cerebral hemispheres (*vide* microscopical specimens). *Rhythmical repetitions of one movement and an irregular succession of different movements* have thus been observed. So that the experimental "chorea" differs from and resembles both the "chorea" of animals and that of man. Some more clinical symptoms due to capillary embolism remain to be recorded.

6. Absolute paralysis of the right foreleg was observed in one guinea-pig, and there was also absolute paralysis of the whole of the right side of the face. No reflex action could be got in any part of these paralysed areas, though sensation appeared to be intact. In this case starch granules were found blocking an arteriole in the cervical cord, and I believe this embolism was the cause of the flaccidity and palsy of the foreleg. The emboli were freely scattered over the brain, and I regard the facial palsy as being due to total anæmia of the facial nucleus as the result of embolism; the facial nuclei are homologous with the nuclei in the spinal cord. These observations show that paralysis of spinal order may be due to embolism of the vessels of the spinal cord. Some cases of myelitis and infantile paralysis may have such a mode of origin.

7. Perfect left hemiplegia of cerebral type was observed in a cat after the injection upwards into the right internal carotid. Death followed in two days, and the autopsy showed that the larger vessels were perfectly free from emboli, which were limited to the smallest arterioles, if not to the capillaries alone (*vide* sections). (In this cat there was, in addition, absolute paralysis of the right pupil.)

8. The respiration was variously affected; in some experiments the animals clearly died from paralysis of respiration whilst under the influence of the anæsthetic, but no doubt as the result of embolism of the medulla oblongata.

9. The pulse varied much; the heart's action was sometimes markedly slowed; at others greatly quickened.

10. The temperature in every case was greatly lowered. In the cat and dog it fell from the normal 99° to 94°; in guinea-pigs from 101° (which is about the normal) to 92°—95°. In the hemiplegic cat the temperature of the paralysed side was ·4° colder than the right side. Temperature in rectum 98°, left axilla 95·8°, right axilla 96·2°.

*Generalisation.*—Any symptom or group of symptoms of disease of the nervous system may be caused by capillary embolism. The experimental results above recorded are alone sufficient to defend this position. My investigation also shows that a certain amount of capillary embolism may be present in the convolutions and other parts of the brain without inducing any obvious symptoms during life.

*Anatomy.*—With regard to anatomical appearances there is not much to be said. For the most part the capillary emboli give rise to no changes (in the time during which the animals have lived), the longest duration being ten days. Excepting blocking of the capillaries, nothing abnormal has been seen in the nervous tissues.

In a monkey with perfect cerebral hemiplegia the result of *capillary* embolism, there were areas of capillary congestion of a bluish-red colour, of which Mr. Victor Horsley was kind enough to make a water-colour drawing immediately after removal of the brain. The whole of the right hemisphere was greatly swollen as the specimen still shows, and as is well seen in the water-colour sketch.

In a dog there were minute areas of anæmia surrounded by congestion of capillary vessels in the pia mater of the convex surface. I have to thank Mr. J. J. Lister for a pencil drawing of this dog's brain.

*Pathology.*—I may now pass on to the last division of my paper. In chorea three factors are present, viz. spontaneous movements, inco-ordination of volitional movements and paresis. According to Dr. Gowers ("Paralytic Chorea," 'British Medical Journal,' vol. i, 1881, p. 69) these three phenomena are present in every case, though

one may be present almost to the exclusion of the others. In my experimental observations I have not noticed any appreciable inco-ordination or paresis. I offer the suggestion that the varieties of chorea as observed in ordinary practice are dependent upon the seat of the lesion giving rise to the chorea. It may be that where the inco-ordination is the most marked feature that the seat of the lesion is in another part of the motor tract to that which gives rise to spontaneous movements. The seat of the lesion may possibly be the same whether paresis or clonic spasm is the most marked feature. At least paresis and clonic spasm appear to be closely related to each other. I am not prepared to go so far as to say positively that the *more* involuntary movement the *less* paresis, and conversely. It is possible that what holds good in this respect of the grey matter of the spinal cord does not hold good of the grey matter of the brain.

There is a marked contrast between the "chorea" of animals and that of man. Hughlings Jackson, Broadbent, Bastian, Tuckwell, and Todd, with others, have strongly advocated the cerebral origin of the disease in man for reasons which appear to me to be unanswerable.[1]

I look on the occurrence of nystagmus, twitchings in the face and muscles of the jaw which were present in my experimental observations as due to disturbance of the functions of the nerve nuclei in the pons which are strictly analogous to the nerve nuclei in the spinal cord.

The "uncontrollable" movements or movements of rotation I regard as due to lesion of parts above the spinal cord, and which may be spoken of as the brain proper.

In none of my observations have I seen clonic spasms which could be set down to lesion of the brain proper; this statement applies to guinea-pigs, rabbits, cats, and dogs.

[1] Broadbent's argument that the reflex actions are never exaggerated in human chorea and therefore the lesion is not spinal is not absolutely true. In some cases of human chorea the reflex actions are exaggerated, and in many cases they are perverted.

Why did not capillary embolism of the motor parts of the brain proper in these animals produce choreoid movements or even clonic spasms? I do not pretend to answer this question, but merely offer some remarks which may possibly throw some light thereon.

The pyramidal tracts in the animals in question are but ill developed. Pitres has shown that in the guinea-pig and rabbit the cortico-medullary fibres stop short at the medulla oblongata; in the cat and dog but a slender strand passes down the opposite lateral column of the spinal cord. Nervous discharges originating in the highest motor centres cannot act on the nuclei in the spinal cord of the animals in question without first passing through other ganglionic centres, whereas in man nervous discharges originating in the highest motor centres can by passing down the well-developed pyramidal tracts act directly on the nerve nuclei in the spinal cord.

The "forced" movements observed in my experiments must obviously have been due to nervous discharges originating simultaneously in a large mass of ganglionic tissue situate in one side of the brain; these nervous discharges were doubtless due to the presence of emboli in the vessels of the brain. Trousseau has spoken of "forced" movements under the name of chorea saltatoria. As to what part of the brain was concerned in the production of these "forced" movements I am unable to say, for the emboli were widely distributed.

I suggest that all involuntary movements are dependent —physiologically—on an essential similar nervous discharge; the variety of the involuntary movement must be explained on such grounds as difference in the site of the lesion, and possibly to combinations of lesions and the like.

Capillary emboli may cause clonic spasm either after the manner of an irritant or as the result of a defective nutrition of the grey matter supplied by the obstructed capillaries. I submit that the latter view is the more plausible one. According to my observation the movements

tended to increase in frequency. The first indication of loss of nutrition would, no doubt, be a loss of control. A negative condition—loss of nutrition—would thus be capable of causing a positive condition—clonic spasm or tonic spasm. The difference between clonic and tonic spasm is probably one of degree.

The state of innutrition of the motor centres on which I believe chorea depends may be brought about by many causes. Bastian's theory of thrombosis of minute vessels and Dickinson's theory of hyperæmia and exudation receive support from the results of my experiments.

My observations clearly show that the effects of capillary embolism may be entirely recovered from; and this is an argument in favour of the theory that chorea can be caused by capillary embolism.

From my experience at the Hospital for Sick Children and elsewhere I have been brought much into contact with cases of chorea. I have been struck with the remarkable association of paralysis with involuntary movement.

The facts of "paralytic chorea" as observed by Hughlings Jackson, Broadbent, Gowers, and as I can myself bear witness to, are very suggestive of the pathology of chorea.

A good interpretation, and probably a correct one, is to regard the paralysis as the expression of a profounder damage of nutrition of motor centres than gives rise to chorea. Chauveau has recorded a case of paralysis of the extensors with chorea of the flexors of a leg in a puppy. Ferrier has made a generalisation to the effect that the extensors and abductors generally are weaker than the flexors and adductors. Apply this to Chauveau's puppy, and we may suppose that there was the same degree of innutrition in the nerve nuclei energising the puppy's diseased leg, but that degree of innutrition caused paralysis of the weaker extensors and chorea of the stronger flexors.

"*Chorea*" *in animals as described by authors.*—Hughlings Jackson, Gowers, Onimus, and others have shown

that chorea in the dog continues after section immediately below the medulla oblongata. This circumstance, together with the fact of the discovery of anatomical alterations only in the spinal cord, points to the conclusion that the chorea is dependent on disturbance of the functions of the spinal cord. My experiments support these conclusions.

Anacker has described "chorea" in the cat and cow. In the cat the movements were limited to the head and neck and eyes (horizontal nystagmus); the affection came on whilst the cat was suckling and ceased after the completion of suckling. In the cow the affection supervened on calving; the movements were limited to rhythmical jerkings of the head from side to side.

Chauveau has observed "chorea" in the female puppy; in one case there was paralysis and atrophy of the right foreleg, which was followed by chorea; another case I have mentioned above. Hughlings Jackson states very clearly that in the dog "chorea" consists in a rapid repetition of one movement rather than in a succession of different movements.

I introduce the above facts in order to show that the term chorea is a very elastic one. I think it important that we should have a class, called by no matter what name, which shall include every species of involuntary movement. Clonic spasm is easily separated off from tonic spasm, at all events theoretically, and for the most part practically. The large subclass of involuntary movements—clonic spasm—contains many varieties such as simple twitch, rhythmical twitchings, the slow succession of movements of what may be termed typical chorea, the movements of disseminated sclerosis, and so forth.

*Literature of experimental capillary embolism.*—The literature of the subject of artificial capillary embolism is meagre. Panum, Prevost, and Cotard and others have published works on the subject of experimental embolism, but they admittedly occluded much larger vessels than capillaries.

Feltz ('Embolies Capillaires,' 1870) used emboli fine enough to penetrate to the capillaries, but he used very large quantities of such materials as pus and cancerous particles which could hardly be regarded as innocuous substances. Moreover, he was chiefly at pains to prove that capillary embolism of the brain could cause sudden death. My experiments leave no doubt on that point.

*Concluding remarks on chorea.*—Kirkes, Tuckwell, Hughlings Jackson, Broadbent, and Bastian have supported the doctrine of blockage of minute vessels as the cause of *some* cases of chorea, and although some post-mortem evidence has been forthcoming tending to support this view, yet such evidence must always be open to objections. The vascular obstruction may not have resulted from embolism, or it may have been the consequence of the pathological condition on which the chorea depended, and other possibilities suggest themselves. Experimental evidence appears to me to be free from objections of the kind just mentioned.

It may be argued that though I have succeeded in proving that movements indistinguishable from those of chorea can be caused by capillary embolism, yet I have not yet shown that actual chorea can be so caused. It may be urged that it is necessary that the animal shall live, and that the movements shall pass away. The last argument is futile, for I have shown that animals affected with rhythmical movements, with movements of rotation and with simple twitchings do live and recover, and there is no doubt that the movements are in some way dependent on the artificial embolism. I may add that I have monkeys in my possession at the present time which are alive and in good general health three weeks after the induction of capillary embolism of one side of their brains. The collapse which results from embolism of the pons and medulla is the difficulty which cannot be overcome when we wish to cause embolism of the upper part of the spinal cord. I have made an attempt to cause embolism of the

lumbar part of the spinal cord and have succeeded, but then the emboli also get into the arteries of the viscera and cause death in a day. (In this attempt the starchy fluid was injected by means of a catheter from the femoral artery. Complete palsy resulted, but at the upper boundary of the palsy twitchings were present during the time the animal lived. In a second attempt where less starch was injected no nervous symptoms followed and none of the starch could be found in the spinal cord.)

That the twitchings and movements are not dependent on the presence of emboli in the capillaries of the muscles and nerves is apparently proved by the fact that an injection of a large or small quantity of starch into the femoral artery distally may be made without any phenomena at all being observed.

My future efforts will be directed to the investigation of the question whether chorea can be caused by capillary embolism of the cerebral hemispheres of monkeys as being animals the nervous system of which is the nearest approach to that of man.

(For report of the discussion on this paper see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 449.)

# FATTY TUMOURS.

BY

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LIPOMATA are morbid growths which occur so frequently, are so well known, and apparently possess so little scientific interest, that it may with reason be asked, What can there be that is novel to write concerning them?

For the most part these tumours are to be regarded as overgrowths of the subcutaneous tissue, and as such are exceedingly prone to occur on the trunk and proximal segment of the limbs. These examples are not objects for consideration in this paper, but the rarer kinds—those which occur in unusual situations—will afford much scope for speculation.

The first case which excited my interest in the question was that of an old man who, some years previously, had a ventral hernia, through which a small piece of omentum protruded. This had never caused the patient any inconvenience until a few months before he applied to the surgeon, when he observed that the tumour had commenced to grow and had at that time attained the

size of an orange. Later on the man died from intercurrent disease, and an opportunity was afforded for examining the tumour. It was found to communicate with the interior of the abdomen by an exceedingly narrow pedicle, but was uniformly adherent to the subcutaneous tissue of the abdominal wall, from which adhesions it derived its nutrition; the vessels in the pedicle were very few and small. In this case it was obvious that the small piece of omentum originally protruded had engrafted itself on to the subcutaneous tissue of the abdomen, and acquiring new growth had become in fact a "tumour" in the true sense of the word, viz. *it had acquired independence.*

Examples of fatty tumours growing in rare situations will now be described.

In the Winter Session of 1883 I removed the biceps cubiti muscle of an old woman, a dissecting-room subject, the upper part of which had become metamorphosed into a fatty tumour, lobulated as these growths usually are when they occur in subcutaneous tissue. It was not an example of fatty degeneration of muscular tissue such as one sees in cases where muscles are put out of use by infantile paralysis, or injury to the dominant nerve, but a genuine lobulated lipoma. It seems to me that possibly this muscle had been injured, the damaged part had retrograded into fat, assumed an autonomy and grown into a tumour. (Plate IV, fig. 3.)

Professor Turner has recorded a similar growth between the greater and lesser pectorals. Sir James Paget refers to one in the museum of the College of Surgeons connected with the heart of a sheep, and M. Cauchois reported in the 'Gazette des Hôpitaux,' July 5th, 1883, the removal of a large fatty tumour from beneath the tongue.

The information afforded by the examination of the tumour occupying the biceps muscle enables me to decide accurately with regard to a fatty tumour removed by Mr. Pearce Gould from the clavicle of a child. The growth

was situated beneath the sterno-mastoid muscle, and was so firmly attached to the periosteum of the clavicle that a portion of the membrane was removed in extirpating the growth. After removal, that portion of the tumour which rested on the clavicle was found to contain a very thin sheet of striated muscle-fibre, and the inference to be gathered from this fact is—that we have in this case to do with one of those abnormal muscles for which the clavicle is famous; the muscular tissue had undergone degeneration and formed a fatty mass, which as the child grew acquired sufficient size to attract attention, and thus came into the domain of tumours and the hands of the surgeon. It is represented in Plate IV, fig. 4.

As a case supporting the view here advocated, I will refer to an admirably reported case in 'Path. Soc. Trans.,' vol. xxviii, p. 221, by Mr. Butlin. A fatty tumour was removed from the leg of a girl, aged 7 years, by Mr. Thomas Smith. It was first noticed when the child was a year old, the time at which she began to walk. The tumour occupied the upper and back part of the leg, a little below the knee. When removed it was found that the growth lay among the deep muscles of the leg, and passed between the tibia and fibula, thrusting the interosseous membrane in front of it.

The tumour, which was of the size of a fœtal head, was composed of fibrous and adipose tissue in equal proportions. It was enclosed in a capsule, and the various fatty lobules were surrounded by broad trabeculæ of fibrous tissue. In the middle of many of these fibrous trabeculæ thin reddish bands or fibres could be seen which afterwards turned out to be striated muscle-fibre. The fibres ran in various directions throughout all parts of the tumour, but were most abundant in a longitudinal direction and towards the anterior part of the mass.

Mr. Butlin concludes that most of the muscular fibre was enclosed within the tumour during its growth and was not of new formation.

Here, as in the instance of the biceps, and Mr. Gould's

case previously described, it may reasonably be supposed that we have to do with an aberrant muscular slip which has undergone fatty degeneration, and the retrograded elements of which have assumed an independence and grown into a tumour. The curious bands, capsule, and trabeculæ recall strongly the arrangement of connective tissue between the fasciculi of an ordinary limb muscle.

In the 'Path. Soc. Trans,' vol. xii, p. 148, Mr. Thomas Smith records a case of especial interest on account of its bearing on this question of muscular tissue degenerating into fat. Sir James Paget had removed a pedunculated fibro-muscular tumour of the uterus. On laying open the growth it was found to contain a cyst and a fatty tumour the size of a pigeon's egg, and to be surrounded by a fibro-cellular capsule.

There can be little doubt that this mass of fat, occurring in a situation usually devoid of fat, is a good example of the doctrine here advanced concerning these fatty tumours in "atypical" situations.

By way of broadly illustrating the question, a few examples from some of the domestic animals are given.

If the scrotum of a bull or ram be examined, it will be found to contain scarcely any fat, practically none. Examine now an ox or a wether sheep, say a year after they have been castrated. The scrotum is composed of an exceedingly thin layer of skin enclosing a large amount of fat, indeed, in the case of the ox it may weigh several pounds. Why is this? The testicle having been removed, the dartoid tissue and the proper coverings of the testis have degenerated into fat which, having no function to fulfil, has grown into the condition of a "tumour." "Hen birds" offer themselves as additional examples. In the early chick, two ovaries and two oviducts may be seen, but in the adult bird the right ovary has disappeared; often the oviduct shares the same fate, but frequently it is represented by a small duct about half an inch in length connected with the cloaca. This remnant may exhibit two forms of degeneration: (*a*) The rarer is a

cystic condition; but more commonly one finds (*b*) a small fatty tumour developed on its upper extremity. This occurs most constantly in tame birds as fowls and pigeons. (Plate IV, fig. 2.)

It is well known that lipomata occasionally develop in connection with the alimentary canal, particularly in horses and oxen. Two remarkable tumours of this sort have come under my notice. They were found *loose* in the peritoneal cavity of a mare which was killed at the Zoological Gardens for the purpose of feeding the animals. The larger of the two measures four inches by three, the smaller three inches by two and a half, both are covered with a layer of serous membrane, and after careful examination no trace of a pedicle could be discovered.

On section they present the granular appearance so characteristic of omental fat.

The only opinion to offer concerning these masses is that they represent overgrown "appendices epiploicæ" which hung suspended to the gut by an extremely narrow pedicle, and that during the struggles of the animal in the process of killing they became detached, for it is improbable and inconsistent to suppose that they lived and grew loose in the peritoneal cavity.

*Inflammatory new-formations may degenerate into fat and become veritable fatty tumours.*

In the museum of the Royal College of Surgeons is a specimen in the general pathology series, No. 196[a] the gift of Dr. Norman Moore. "It consists of a section of a mass of fat which surrounded a portion of the ureter, in which a calculus was impacted. Near the centre of the section a small portion of the wall of the ureter is seen, upon which the calculus rested; bands of fibrous tissue pass from it into the fatty tissue. The mass formed a distinct tumour. It was probably produced by fatty degeneration of newly-formed fibrous tissue (inflammatory new-formation) occasioned by the irritation of the calculus." The above account is taken from the catalogue.

The museum of the Middlesex Hospital contains a

specimen showing a rectum and anus, the seat of a stricture the result of syphilis. The lower portion of the bowel, uterus, and vagina are embedded in a mass of fat measuring three inches from before backward. This mass of fat is divided and subdivided by bands of fibrous tissue, which in places give it a reticulated appearance. Sinuses lead from the gut to open around the margin of the anus. This seems to be a specimen of the same nature as the preceding one, and may be explained in this way. In the early stage of the stricture a certain amount of inflammation was present, which led to exudation of inflammatory products; these in their turn retrograded into fat, which later on, irritated by the stricture and the sinuses leading from the bowel, grew into a tumour. It must be remembered, however, that this retrogression of inflammatory new-formations completely disguises all traces of the original inflammation.

Dr. J. K. Fowler first drew my attention to the very frequent presence of a large collection of fat around the bowel in cases of long standing stricture, having himself observed it in very many instances.

Mr. T. W. Nunn reported to the Pathological Society, for Mr. Worthington (vide vol. xv, p. 100), an example of a fatty tumour surrounding the rectum, and on carefully going into the details of the case there can be little doubt that it was of precisely the same nature as the two cases just recounted. It was associated with old standing disease of the uterus, and thus serves as a striking confirmation of Dr. Fowler's observations. As a curious and valuable case in support of the view here advanced as to the probable origin of these collections of fat, the following may be used as an illustration.

M. Paul Berger relates, in the 'Gazette des Hôpitaux' of November 15th, 1883, that a man was admitted into La Charité with a salivary calculus impacted in Steno's duct. The irritation had caused hypertrophy of the pad of fat lying between the masseter and buccinator muscles, known as Bichat's *boule graisseuse*.

In these cases of fatty degeneration of inflammatory new-formations a large quantity of fibrous tissue is present which forms alveoli containing the adipose matter.

Attention must now be invited to another mode by which fatty tumours may arise, *i. e., by the degeneration of the soft parts of parasitic fœtuses.*

The museum of the Middlesex Hospital possesses a fatty tumour, containing a shapeless mass of bone, which was removed from the buttock of a man by Mr. Henry Morris, November, 1876. Bone in a fatty tumour is by no means unknown, indeed, several cases have been recorded, but it is of rare occurrence.

The question which now suggests itself is this :—Are these tumours containing bone to be regarded as "lipomata" in the ordinary sense of the term? Certainly not. They are examples of immature fœtuses attached to the trunk of a perfect individual, or, as they are often called, "parasitic fœtuses," in contradistinction to the autosite or mature being who is the unfortunate bearer of one.

The explanation would be that the soft parts of the attached fœtus undergo fatty metamorphosis, but the bone remains unchanged. These may continue quiescent throughout a long life, but from some cause or other, we know not what, may suddenly take on active growth, in the same way as the piece of omentum in the first case considered.

As an undoubted example of fatty tumours arising in attached fœtuses, reference should be made to the Teratological Catalogue of the College of Surgeons Museum, No. 132.

This specimen consists of the head of a human fœtus with a large lobulated vascular tumour, the remains of a second fœtus, growing from the median fissure of the palate. The bulk of the tumour is made up of fat. (Full details will be found in the catalogue.)

Mr. Butlin refers to a case described by Arnold. A child survived its birth six days with a large tumour filling

its pharynx and mouth. It was a lipoma, inasmuch as it consisted chiefly of fat, but it contained also fibrous tissue, cartilage, cysts, and even striated muscle-fibres ('St. Barth. Hosp. Rep.,' vol. xiii). A very remarkable case is recorded in 'Path. Soc. Trans.,' vol. xxxiii, p. 287, by Mr. Frederick Treves. An instance of fatty degeneration of an attached fœtus similar to Arnold's case came under my notice this year, but the parents refused to give me the growth.

It will now be important to ascertain, if possible, on what this fatty metamorphosis depends.

An interesting fact is pointed out by Otto in his 'Compendium of Human and Comparative Pathological Anatomy' (South's Translation, 1831) to the effect that parts devoid of nerves are exceedingly prone to undergo fatty change, and by way of example he cites cases of parasitic fœtuses which as a rule are without nerves, and contain a considerable quantity of fat in lieu of more important tissue, such as muscle. By way of illustrating the relation between absence of nerves and presence of fat I will detail a remarkable case which came under my own observation.

Early in 1883 Mr. Edgar Nicholson obtained for me a fœtus with the following history :—The mother, a healthy woman who had passed successfully through several pregnancies, and was again in the fifth month of gestation, fell down six stairs on to her stomach, with her legs doubled beneath her. Stunned by the fall she lay in that position until help arrived. After remaining in bed for a few days she felt well again, but never felt the child move about as in previous pregnancies, and she feared it was dead. At the eighth month the child was born, lived for four or five hours, and then died.

On examining the body it was found that whereas all parts above the umbilicus presented the appearance and size of a child of eight months of intra-uterine life, the parts below were those of a fœtus about the fifth or sixth month of gestation.

Looking to the spinal column it was found to present the sac of a spina bifida at the first lumbar vertebra, and at this point the canal and column suddenly ended; no trace of the remaining lumbar, sacral, or coccygeal vertebræ could be found. The ossa innominata were fused along their vertebral borders, the ureters were dilated and the kidneys cystic. No trace of the lumbar, sacral, coccygeal, or sympathetic nerves could be detected.

On reflecting the skin of the legs, which was very thin, I was surprised to find that they consisted of bones corresponding in their degree of development to about the fifth month of gestation, bone and cartilage being present in due proportion, but all other tissues, except a few blood-vessels, being represented by fat; indeed, these legs would be best described as *fatty tumours in the shape of lower limbs, containing bone and cartilage.*

The explanation seems to be that when the woman fell, she fractured the child's spine, destroyed the continuity of the spinal cord, and deprived the limbs of their nerve supply, which, in consequence, *degenerated into adipose tissue.* (Plate IV, fig. 1.)

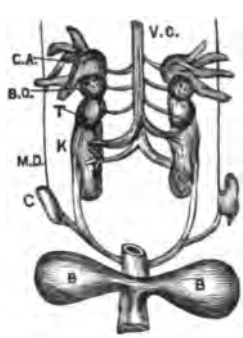
In spite of this evidence it may be still fairly argued that we have no satisfactory evidence that these attached fœtuses ever had any important tissues to suffer degradation, and as one cannot say positively that they had, the evidence on that point remains more or less circumstantial. But I shall now proceed to detail a case where the process has been traced from the perfect tissue to the fatty condition, and, what is still more important, the tissue in question occasionally persists in its perfect form.

Such evidence is afforded by:

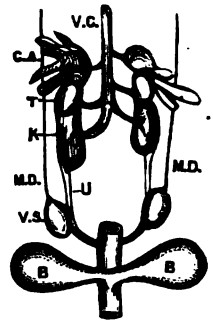
### *The Fat Body of Frogs and Toads.*

If the abdomen of a frog, *Rana temporaria,* be opened and the alimentary canal with the liver be removed, the reproductive organs will then come into view. If the frog

be a male the following disposition of parts will be observed:




Toad.




Frog.

T. The testes. A pair of yellow oval bodies, dotted with black specks, usually one fourth of an inch long, lying on the ventral surface of the kidneys. From these issue the vasa efferentia, about ten or twelve in number, which pass into the median border of the corresponding kidney. The spermatozoa pass from these into the tubules of the kidney to escape by the ureter, U, which in the frog serves not only as the main duct of the kidney but also as a vas deferens. Just before the ureters enter the cloaca, C, they form pouch-like dilatations known as the vesiculæ seminales. Passing from the summit of these receptacles in the male frog is a thin streak representing the Müllerian duct, which reaches as far forward as the root of the lung, M.D. Lying on the anterior extremity of the testis and sometimes connected with it is a mass of fat with three or four finger-like processes. This collection of fat is known as the "fat body" or corpus adiposum, C.A.

Turning now to the female frog the following arrangement is seen:

O. The ovaries. A pair of black masses lying in the folds of the peritoneum in front of the kidneys, their surface dotted with ova, and varying in size, shape, and colour according to the time of year.

M.D. The oviducts. A pair of convoluted tubes commencing at the root of the lung with funnel-shaped orifices and ending posteriorly in the cloaca, the genital ducts in the female being quite distinct from the ureters.

Lying on the anterior end of the ovary and connected with it is a corpus adiposum exactly corresponding to that on the testis of the male.

It is to this fat body that attention is now invited, for its history affords us a clear and instructive example of the process by which the rarer kinds of lipomata we have been considering indubitably arise.

The origin of the body in question has attracted the attention of some careful observers. Von Wittich's researches clearly show that the fat body and testis are developed from the genital ridge in the embryo, but the anterior part undergoes fatty degeneration, whereas the posterior part develops into a testis. This means that that portion of the genital ridge which under certain conditions becomes part of the ovary is represented in adult frogs by fat. There can be very little doubt on this score, for if anyone has the patience to examine say fifty frogs, he will be pretty certain to find one or more frogs in which the fat body is replaced by an ovary in addition to the testis, so that the frog possesses in fact an ovo-testis.

The common toad, *Bufo vulgaris*, affords excellent and important evidence on this head.

In the male toad, wedged in between the testis and the fat body, is a third structure, usually referred to as "Bidder's organ" (see woodcut on page 302). Careful inquiry by numerous competent observers has settled, on microscopical and embryological grounds, that "Bidder's organ" is really a persistent portion of the ovary which has not been involved in the general fatty metamorphosis of the anterior end of the genital ridge.

There is another fact which serves to demonstrate the ovarian nature of "Bidder's organ," and to which due consideration has not been given. In the toad the Mülle-

rian ducts, M.D., are always more obvious than in the frog, and the careful examination of several scores of toads has convinced me that the amount of development of the ducts in question corresponds to the size of Bidder's organ. That is, as a rule, if these ducts are large Bidder's organs are well developed and *vice versâ*. Also in those specimens of *Rana temporaria* possessing an ovo-testis which I have examined—and at least five examples have come under my notice in examining 250 frogs kindly collected for me by my brother—the Müllerian ducts (oviducts) have been developed in corresponding proportion. Hence it would seem that as the abnormal ovary approaches more and more to a mature condition so do the accessory ducts assume a functional size.

Now and then a frog may be found in whom the metamorphosis has exceeded its normal limits so as to convert a *part or even the whole of the testis into fat.*

A similar condition may affect the female, whereby a portion of the entire ovary of one side (according to my observations usually the right one) may degenerate into adipose tissue.

These facts go to show that in Amphibians (but not confined to this group) there is a tendency on the part of the anterior extremity of the genital ridge, instead of developing into an important tissue, to undergo conversion or degeneration into fat which now and then exceeds its general limits. *Hence, nearly every frog possesses as part of its internal anatomy "a fatty tumour."*

It is important also to notice that this metamorphosed tissue behaves itself exactly like a tumour: "it has a growth independent of the rest of the body."

For if the anterior end of the genital ridge develops into ovarian tissue it remains of a proper size, and I have never seen it exceed normal limits, but when represented by fat *it grows and increases in size,* for it has no function to keep it in check.

Lastly, it is important to note that, in common with fatty tumours affecting man, general emaciation has no

effect upon these fat bodies of frogs; indeed, so far as my dissections go, the thinnest frogs and toads have the largest and best developed fat bodies. In this respect they differ from camels, in whose humps these collections of fat diminish towards the end of a long and toilsome journey.

Another class now awaits consideration, viz. "Dermoid Cysts."

It is a well-established fact that these cysts may be made up of the most complex structures, including in one tumour skin with its glands, hair, teeth, bone, muscular, and in rare cases nervous tissue. Fat also enters largely into the composition of these growths. If the facts recorded in the first part of this paper be correctly interpreted, then there can be little reasonable doubt that in the early stage of these dermoid cysts, most of the complex tissues of the body may enter into their composition, but lacking function, may later undergo fatty degeneration, as in the examples of parasitic fœtuses previously considered. The explanation is a simple one and suggests a probable mode of accounting for the origin of fat in these very remarkable formations.

Lastly, a few words must be written about the "Lipoma arborescens" occasionally found in the neighbourhood of joints affected with chronic disease. The only example I have seen is the very beautiful specimen (No. 327 in the Catalogue of the Museum of the College of Surgeons) from whence the following account is extracted:

"A section of the condyle of a femur, around the articular margin of which there is a crowd of small oval and branched growths, of fibrous and fatty structure, each covered by a shining membrane, like a reflection of synovial membrane, and all attached by long and slender pedicles. Their shapes and sizes are various; some are like branching threads; others are nearly cylindrical; others flattened and lanceolate. They are examples of the "Lipoma arborescens" of Müller. The articular cartilage has been removed from the front of the condyle, and the exposed bone is hard and polished, with an

appearance of gouty deposit on it; elsewhere the cartilage is thin."

It seems to me that in this specimen we have to deal with a case in which villous growths sprouted from an inflamed synovial membrane, but their nutrition was interfered with, on account of their long and slender pedicles; hence diminished nutrition of the little growths led to their fatty degeneration.

It is a fact well recognised in pathology, that, as a rule, growths with long and slender stalks rarely attain any important size and often suffer necrosis or detachment in consequence; hence it is not propounding anything far-fetched or fanciful to suggest that these minute fatty tumours arise by *degeneration of the villous processes which frequently infest the synovial membranes of joints in certain chronic diseases.*

Mr. Butlin in his paper on "Fatty Tumours in Infancy and Childhood" ('Barth. Hosp. Rep.,' vol. xiii) has put forward an opinion, that the occurrence of fat in many of these tumours is the result of secondary changes, and refers to a case reported by Weber, in which a tumour consisted of telangiectasis, fat, and fibrous tissue; it was supposed that the fat and fibrous tissue resulted from secondary changes occurring in a nævus.

In writing on the subject of fatty tumours in his 'Surgical Pathology' Sir James Paget says: "Respecting the causes of these tumours few things can be more obscure, nearly all knowledge on this point is negative."

May one venture to hope that the few facts related in this paper will go a little way towards dispersing some of this darkness.

The substance of the paper amounts to this:

*Any of the soft tissues of the body, normal or pathological, may degenerate into fat, and this retrograded tissue does, in some instances, assume an autonomy and grow into a fatty tumour.*

A list of references to interesting examples of fatty

tumours, whose details have a bearing on the views advocated in this paper, is appended.

*Fatty Tumours in Unusual Situations, &c.*

*Bone.*—Attached to ischium. Mr. T. Smith, 'Path. Soc. Trans.,' vol. xvii, p. 286. To the neck of the radius. Mr. T. Smith, 'Path. Soc. Trans.,' vol. xix, p. 344. Attached to the clavicle. Mr. Pearce Gould, p. 294 of this paper. To coccyx. Mr. T. Smith, 'Path. Soc. Trans.,' vol. xxi, p. 334.

*Nervous System.*—A fatty tumour growing from the superior peduncles of the cerebellum. 'Catalogue of Museum,' Middlesex Hospital, No. 834. From the cranial dura mater. Mr. Sibley, 'Path. Soc. Trans.,' vol. vii, p. 1. In the interior of the spinal canal near the seventh cervical and first dorsal vertebræ. 'Path. Soc. Trans.,' vol. iii, p. 248, by Mr. Obré. Median nerve in the palm of the hand. Mr. Pearce Gould, 'Catalogue of Museum,' Middlesex Hospital, No. 2191. Fatty tumours on nerves. Virchow's 'Archiv,' Bd. xv, s. 61. Lipoma of sacrum connected with the spinal membranes. Mr. Athol Johnson, 'Path. Soc. Trans.' vol. viii, p. 16.

*In Muscles.*—In the heart of a sheep. Museum, College of Surgeons. Between the greater and lesser pectorals. Prof. Turner, quoted in Paget's 'Surgical Pathology.' In the tongue simulating ranula. Dr. F. Churchill, 'Path Soc. Trans.,' vol. xxiii, p. 234. Involving the biceps (Author). Beneath the tongue. M. Cauchois, 'Gaz. des Hôp.,' July 7th, 1883. Involving the muscles of the calf. Mr. T. Smith's case, reported in 'Path. Soc. Trans.,' vol. xxviii, p. 221. In a fibro-myoma of the uterus. Mr. T. Smith, 'Path. Soc. Trans.,' vol. xii, p. 148. Beneath occipito-frontalis; also one attached to the complexus, Mid. Hosp. Museum.

*Intestines.*—Of an ox. 'Catalogue of the Museum,'

College of Surgeons, No. 324. A fatty tumour projecting into the cavity of the gut at the angle of junction of the two segments of the ileo-cæcal valve. Prof. Turner, Paget's 'Surgical Pathology.' Attached to the mesentery. Sir Spencer Wells, 'Path. Soc. Trans.,' vol. xix, p. 243. In the abdomen. Weighing 29¾ lbs. Mr. Pick, 'Path. Soc. Trans.,' vol. xx, p. 337. In abdomen; containing osseous tissue. Dr. Dreschfeld, 'Path. Soc. Trans.,' vol. xxxi, p. 287.

*Throat.*—In pharynx and larynx. Mr. Holt, 'Path. Soc. Trans.,' vol. v, p. 123. In pharynx. Dr. Frederick Taylor, 'Path. Soc. Trans.,' vol. xxviii, p. 216. The right arytæno-epiglottidean fold. Mr. Sydney Jones, 'Path. Soc. Trans.,' vol. xxxii, p. 243.

*External Organs of Generation.*—To the scrotum. Mr. Henry Gray, 'Path. Soc. Trans.,' vol. vi, p. 230. To the labium. Unreported. A case of the author of this paper.

*Lower Limb.*—Sole of foot. Mr. Gay, 'Path. Soc. Trans,' vol. xiv, p. 243.

*Fatty Tumours in Parasitic Fœtuses.*—Attached to the head of the autosite. Museum, College of Surgeons. 'Teratological Catalogue,' No. 131. Attached to coccyx. Mr. Frederick Treves, 'Path. Soc. Trans.,' vol. xxxiii, p. 287. Attached to sacrum, lipome calcifié congénital. Dr. L. Briolle, 'Gaz. des Hôpitaux,' Jan. 23rd, 1883. Attached to median line of palate and filling the pharynx and mouth. Arnold's case referred to by Mr. Butlin in 'Barth. Hosp. Rep.,' vol. xiii. Many examples are given by Braune, Geschwülste der Kreuzbeingegend.

(For a report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 456.)

## DESCRIPTION OF PLATE IV.

(Fatty Tumours, by J. BLAND SUTTON, F.R.C.S.)

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FIG. 1.—The lower limbs of the fœtus described in the paper, dissected to show the degeneration of the tissues into fat. F. Femur. A. T. Adipose tissue. T. Tibia.

FIG. 2.—The left oviduct of a pigeon. The rudimentary right oviduct is shown with a fatty tumour developed on its summit.

FIG. 3.—Biceps muscle from an old woman. The upper part is occupied by a fatty tumour.

FIG. 4.—A clavicle with a fatty tumour attached. Mr. Pearce Gould's case.

Fig. 1.

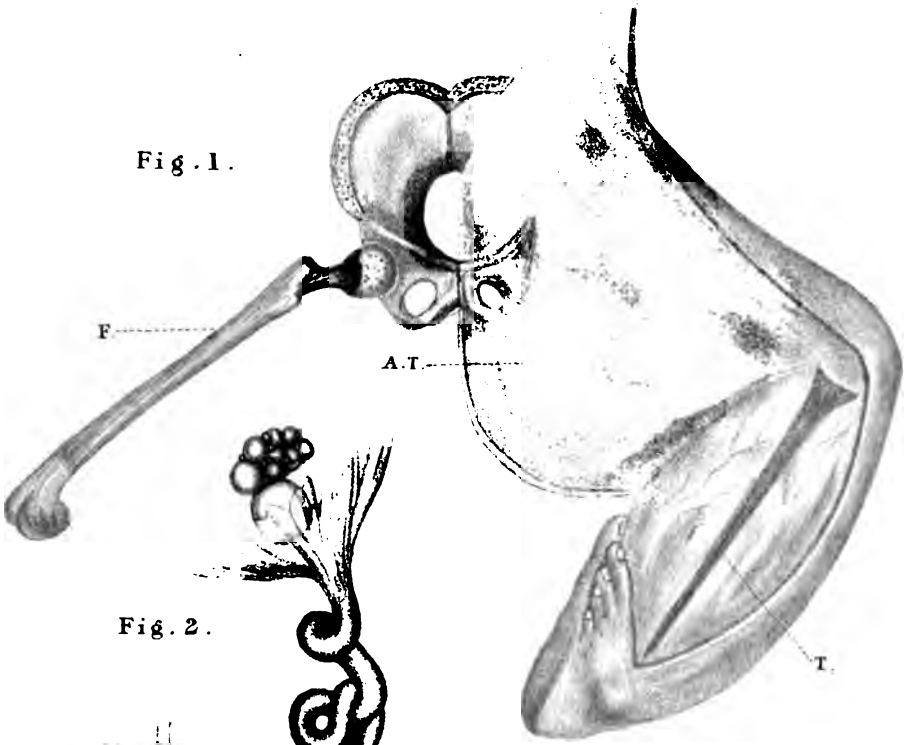
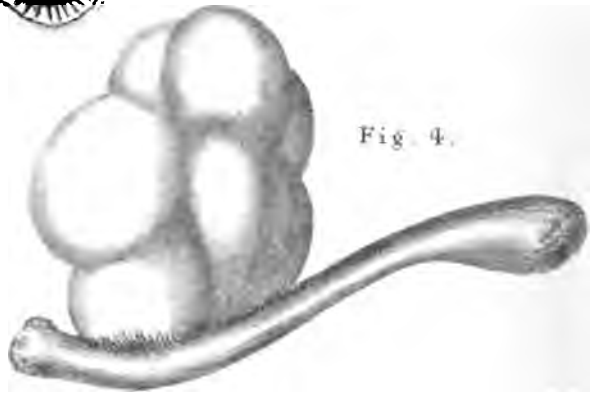



Fig. 2.

Fig. 3.

Fig. 4.



Mintern Bros. lith.

# ON A CASE

OF VERY LARGE

# LYMPHO-SARCOMATOUS TUMOUR OF THE TONGUE.

BY

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Received April 14th—Read June 9th, 1885.

THE case which forms the basis of my present communication was under my care about four years ago. The entire tongue of a young man, only twenty-two, was then removed on account of a tumour which had been slowly growing for more than half his life, and which had at length attained such a size that it impeded swallowing and even threatened suffocation. I have purposely allowed a considerable period to elapse before submitting the case to the consideration of the Society, because I wished to have the evidence afforded by time as to the true character of the growth. Two years had passed by, and as the patient remained quite well, I had begun to hope that we might assume it as probable that the tumour was of an innocent character. I am sorry to say, however, that after this long interval these hopes have been disappointed.

The whole mass weighed after removal seven ounces. It was by far the largest tumour of the tongue that I have ever seen removed, and I believe the largest on record.

The subject of the case was a medical student, aged, at the time of the operation, twenty-two years. His father was a medical man, and the conditions had, consequently, been carefully watched from the first. No growth had been observed, and no morbid condition of the organ noticed, during the first few years of life, and his father having had occasion to make applications to the tonsils in childhood, felt sure that he should have seen it, had anything been present.

The first symptom that was observed was at about the age of ten, and consisted of a swelling in the left side of the organ, with a rough papillary growth on its posterior surface. Without causing any inconvenience except from its size, the tumour continued to increase, and when, in the latter part of 1880, I first saw the patient, the organ was fixed in the mouth by its mere bulk, and speech was difficult. At this time the tumour consisted of a large rounded mass, deeply embedded in the tongue, and wedging itself against the sides of the lower jaw. The tip of the tongue was free, and could be moved on the surface of the tumour, but with this exception the whole of the organ was involved. The tumour could easily be felt externally as a hard mass bulging downwards behind the chin. The mucous membrane of the tongue over the tumour was quite healthy, with the exception of the posterior two thirds on the left side, which presented a coarse papillary growth, not in the least painful, nor ulcerated, but which was continuous with the substance of the tumour beneath. The surface at this part was nodular, like the outside of a mulberry. Had it not been for this growth, which implicated the overlying parts, so moveable was the mucous membrane and the superficial layer of muscular structure on the tumour, that one might have been tempted to hope that it was encapsuled, and might possibly be shelled out.

I had had the advantage of the opinion of Sir James Paget, to whose kindness indeed I had been indebted for having the patient placed under my charge, and I subsequently obtained that of Mr. Savory and several other professional friends.

It was felt by all that the time must come when it would be absolutely necessary to operate, and, encouraged by the long duration of the case, we were all hopeful that the disease was not in any sense malignant. As to its precise character, however, no one ventured a confident opinion. The operation was not performed until nearly a year after I had first seen the patient, and in the interval the growth had increased considerably, and the inconvenience had finally become unbearable.

The operation was done on November 20th, 1881, and I had in it the able assistance of Mr. Waren Tay and Mr. R. W. Parker.

The patient having been put under ether a preliminary tracheotomy was performed. It would have been better to have done this without the anæsthetic, for as soon as insensibility commenced the mass fell back upon the glottis and the patient was in the utmost danger from asphyxia. It was impracticable to draw it forward sufficiently to effect relief, and, after a hurried completion of the tracheotomy, we were obliged to do artificial respiration for some time to restore animation. The patient having well rallied the pharynx was next plugged with sponge, and an exploratory incision was made into the substance of the tumour in order to ascertain whether or not it was encapsuled. Finding that it was not so, that it was very firmly fixed on all sides, and that it was quite impossible to get the finger either beneath or beyond it from the mouth, I at once made an incision through the lip and chin and cut the symphysis of the jaw. Having freed the muscular attachments and widely separated the two halves of the jaw, access was gained to the base of the organ. Having well isolated the mass, partly by scissors and partly by tearing, the wire écraseur was

applied over its base just in front of the epiglottis, and the removal completed. There was no trouble from hæmorrhage.

No difficulty occurred in the after treatment. The jaw united well and a rapid recovery followed.

The drawings (Plate V) give a good idea of the appearance of the growth, both on its surface and in section. It will be seen that the mammillated patch occupies an area larger than a crown-piece, and that its growths are at least a quarter of an inch in height. The tumour itself, as seen in section, was almost globular, and measured two and a half inches across. It completely replaced the substance of the tongue, with the exception of a portion about three fourths of an inch in length at its tip. The posterior two thirds of the growth showed a greyish fibrous structure, which was divided into loculi by white bands. The anterior part was red, not so hard, and much more vascular; but obscurely divided in like manner into lobes. On its surface the growth was everywhere bounded by a tolerably well-marked layer of fibrous tissue. On its surface in many parts, especially in front, were seen what looked like the cut ends of small muscular bundles. There were no cysts, and nothing that was conclusively erectile. The muscular bundles of the front part were inseparably attached to the growth.

One half of the tumour was sent to the College of Surgeons, where it was examined by Mr. Eve, the pathological curator, who also obtained the assistance of Dr. Klein. The other half was examined independently by my eldest son, who had the valuable assistance of Mr. Rickman Godlee in inspecting the microscopic sections. These observers all agreed in reporting that the growth was of the connective-tissue type, and probably a round-celled or lympho-sarcoma. Here and there in its substance muscular fibres, atrophied by pressure, could be demonstrated. A tendency to the development of well-formed fibrous tissue was a strongly characterised feature. The enclosed loculi were of very various sizes and contained cells, which had rela-

tively large nuclei and an ill-defined cell substance. Between the cells a fine reticular tissue was seen in parts.

The much enlarged papillæ were infiltrated with cells, not apparently differing from those in the alveoli beneath.

Between the papillæ in some of the fissures the deep epithelial cells had much enlarged and undergone granular degeneration. Everywhere there was a sharp line of separation between epithelium and corium. (Pl. VII.)

It will be well before attempting to discuss the clinical relationships of this case to give its termination, and also to cite any evidence which can be obtained from the records of other cases more or less resembling it.

After his recovery my patient, who was a gentleman of great pluck, at once returned to his medical studies at Belfast. From time to time I heard of his being quite well, and two years after the operation I heard that he was intending to present himself for examination at our London College, and that I should then have an opportunity of seeing him and hearing how well he could make himself understood. About six months later, however, I had the disappointment of being informed of his death. A growth had very rapidly sprouted up in the floor of his mouth and had in a short time brought about the fatal event, partly by pressure and partly by hæmorrhage. I did not hear of the recurrence until after his death, and there was no post-mortem. I believe that there was no evidence of gland disease.

I am not able to produce either from the records of other surgeons or from my own experience, any case which is an exact parallel to the one which has been described. The following cases, however, in a fragmentary way, some from one aspect, and some from another, seem to throw light upon its probable nature. We may note that we have to deal with a tumour which invaded the substance of the organ in which it was placed; which

began in very early life; which was attended by coarse mammillation of the surface; which continued to grow painlessly and very slowly through a long series of years; which never produced gland disease; which recurred locally after removal, and which under the microscope presented the characters of an infiltrating lympho-sarcoma, with dilatation of blood-vessels.

The first case to which I shall refer is that of a child, aged three, in whom a mammillated growth on the posterior part of the tongue had been present from birth. It had caused no inconvenience but was slowly increasing. I excised it by means of the cautery, and the child, I believe, remains well. It did not go deeply into the substance of the tongue.[1] The method of removal destroyed the specimen for microscopic purposes.

Specimen 1067A in the Museum of the College of Surgeons is the tongue of an infant in whom similar conditions, but on a much larger scale, were present. It was shown by Dr. Hickman, in 1869, at the Pathological Society, and is described in vol. xx of the 'Transactions.' The infant was born with a mammillated tumour on the left side of the dorsum of its tongue as big as a plum stone. It was sessile and extended from the line of the circumvallate papillæ back to the epiglottis. Owing to its peculiar position, rather than its size, it caused death by suffocation within sixteen hours of birth. A committee appointed by the Society reported that it consisted of hypertrophied gland follicles and ducts with an intervening matrix of nucleated connective tissue. Its blood-vessels were large and numerous, but there was no definite erectile tissue.

Mr. Liston has recorded a case in which he operated on a lad, aged nineteen, for a tumour of the tongue which had been present from birth. In some respects this case resembles the ordinary macroglossia of young children, but in others it differs from it, and is suggestive rather of

[1] I possess a coloured sketch illustrating this case. It was produced at the Society's meeting.

a nævoid mole. The tongue filled the mouth and projected over the lips. It was elastic and compressible and its surface was crossed by large venous trunks. The papillæ over it were much enlarged, and granular points were numerous. There was an ulcerated fissure near its centre which frequently bled. Mr. Liston tied the lingual arteries and then ligatured the whole tongue. Unfortunately the patient died of pyæmia.

My next case (Plate VI) is clearly very like Mr. Liston's excepting as to size. Its subject was, when I first saw him in 1872, a lad of twelve. He is now a porter at Woking Junction aged twenty-four, and in good health. The state of his tongue has not materially changed. The portrait shows considerable hypertrophic enlargement of the organ chiefly of its left half, and the surface is covered with mammillated and granular points, just such as Mr. Liston described, and such as were present in the case which is the chief subject of this paper. Some of them were vascular, like parts of a venous nævus. The condition had been noticed soon after birth and it increased somewhat in growth. He was sent to the London Hospital from Farnham when he was twelve, because it had increased somewhat, and with a view to operation. I kept him under observation a few weeks and finding that the condition caused the boy no inconvenience whatever I deferred any interference. My decision has been justified by the fact that he has got no worse. This case seems to differ from that which my paper chiefly concerns in that there is no tendency to continued growth in the substance of the organ. It is very possible, however, that this tendency may yet declare itself.[1]

The next case was a child under the care of Mr. Waren Tay in the London Hospital. The tumour was congenital and deeply placed in the substance of the left half of the

[1] The man who is the subject of this case attended the meeting for inspection. Further examination made it certain that although, as may be seen in Plate VI, the growth appears to involve both halves, it is really limited to the left, and only encroaches on the right by bulging over.

tongue, bulging both below and on the dorsum. The tip of the tongue was free, there was no papillary hypertrophy, and the tumour, although partly solid, consisted chiefly of cysts. I believe the child died after an operation for its removal.

Closely parallel to this, and presenting also features of similarity to my own, is a case which I find narrated by my friend Dr. George Brady, of Sunderland, in the 'Medical Times and Gazette' for 1867. An infant had, at birth, a bluish looking tumour under and in its tongue, which was taken for a cyst. It was punctured freely, and almost fatal bleeding followed. At the age of fourteen Dr. Brady attempted the removal of the tumour, which was then a softish solid. It was sought to excise the growth from under the tongue without removal of the latter, but the tumour was so ill-defined and the bleeding was such that the operation was not perfectly completed. The boy, however, recovered and at the end of several years was still well and without any new growth.

A case which has been recorded, in excellent detail, by Mr. Henry Arnott, in the 'Pathological Transactions' (vol. xxiii), well illustrates the combination of congenital hypertrophy of various different structures in the same organ. The infant was fourteen months old when he was operated on by Mr. Simon in St. Thomas's Hospital. The tongue was of very great size and lolled out between the stretched lips. It had been large from birth and had hindered sucking. The child died of pneumonia three weeks after removal, by the écraseur, of the greater part of the tongue. Mr. Arnott records that "there were present, first, a nævoid affection of the blood-vessels; secondly, a thickening and induration caused by a long-continued sub-inflammatory state; and thirdly, a general enlargement of the lymphatics. Rather large irregular spaces, with very thin walls, and mostly with no visible contents, were met with in every section. Some of these spaces were filled with blood, but for the most part they contained only clear fluid with a few vesicular bodies—swollen

epithelial cells—in contact with their walls, possibly dilated lymphatics."

In none of the cases which I have quoted, excepting my own, was there any proof of the presence of any solid growth which might deserve the name of lympho-sarcoma. In Mr. Brady's case the growth was solid and very possibly of this nature; but it was not so diagnosed, and in Mr. Liston's we have no microscopic examination at all. In several of the others we must remember that the child died after operation in early life and thus no opportunity was afforded for the further developments which took place in mine. The only recorded example of sarcoma, so diagnosed, which I have been able to find, is one published by Professor Jacobi, of New York, and quoted, with valuable criticism and additional facts, by Mr. Butlin in his 'College Lectures.' In this instance the tumour was the size of a hazel-nut at birth, and as big as a walnut when, at three months old, it was removed. It was a firm, rounded, elastic mass, deeply furrowed on its surface. Its section was uniform, excepting that in its centre was a small cyst. Its external portions contained muscular tissue, but its chief structure was that of a spindle-celled sarcoma. There were some round-cells, but not much intercellular substance. The child in this instance was well five months after the operation but nothing more is known of it.

The last case which I shall quote is one published by Mr. Folker, of Hanley. In it a large solid tumour which had been growing for twenty years was successfully removed from the tongue of an adult man. It was diagnosed as "fibrous."

The cases which I have adduced justify, I think, the conclusion that my case ought to rank as one of a group in which hypertrophic structures, present in the tongue at the time of birth, subsequently take on a more or less erratic development. These congenital excesses are probably analogous to moles and nævi of the skin; and in the tongue, as proved in several cases, dilatation of blood-

vessels and of lymphatics is a conspicuous element in the growth. We know that moles of the skin often show vascular as well as papillary and fibrous hypertrophy. All the structures, in irregular proportions in different cases, are involved much as we have noted in the series which I have this evening produced. Moles and nævoid moles of the skin sometimes take on growth tendency, and this may happen at any period of life, and sometimes, as is well known, melanotic sarcoma may occur in them. In some rare instances fibroid hypertrophy in moles shows a definite tendency to recur. I once removed for a young lady a huge pigmented and papillary mole which grew on the pubes and involved the labium. It was impossible to remove the whole. A tendency to subsequent growth was most definite, and second and third operations were required. The subcutaneous fibroid hypertrophy produced a dense hard mass an inch and a half in thickness. The pathological processes displayed in this case were probably very similar to those which occurred in the tongue in the case which I have had the honour to bring before the Society.

In conclusion a few words may be said as to the surgical aspects of the case. It might, perhaps, have been better, as regards the prospects of immunity, if I had operated when the patient first came under my notice. My reasons for delay were, that the growth had already attained such a size that the operation must of necessity be a very formidable one, and that as it seemed highly probable that the tumour was innocent so it was wise to put off the danger as long as possible. Guided by our experience in this instance, I should certainly in any similar case—in which a tumour in the tongue, however apparently innocent, was steadily growing, however slowly—be inclined to advise an early removal. It may be doubted, however, whether a patient in whom such a growth caused neither pain nor inconvenience and had been present for years, would be inclined to submit early to an operation involving the loss of the entire tongue. Such

would have been the conditions under which any surgeon who saw this case before I did would have had to advise.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. i, p. 458.

## DESCRIPTION OF PLATES V, VI, AND VII.

(On a Case of very large Lympho-sarcomatous Tumour of the Tongue, by JONATHAN HUTCHINSON, F.R.S.)

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### PLATE V.

Case of Lympho-sarcomatous Tumour (see p. 311).

FIG. 1.—The tongue, after removal, seen from before.
FIG. 2.—The same seen from the side.
FIG. 3.—A longitudinal section of the tongue from tip to base.

### PLATE VI.

Case of Congenital Unilateral Hypertrophy of Tongue (see p. 317). (A papillary, lymphatic, and nævoid mole.)

### PLATE VII.

Microscopical Sections from Tongue, figured in Plate V. (Drawn by Mr. J. Hutchinson, jun.)

FIG. 1.—Section of the surface of the tongue made through part of two of the large "papillæ" which covered it. The epithelial layer was everywhere well defined, and here presents nothing abnormal; the subjacent layer is very vascular and somewhat infiltrated with lymphoid cells, whilst towards the lower part these show a tendency to be arranged in irregular alveoli.

*a.* Horny layer.
*b.* Rete mucosum.
*c.* Vessels.
*d.* Deep part beginning to show an alveolar arrangement.

FIG. 2.—A section characteristic of the great mass of the tumour, showing large rounded alveoli filled with lymphoid cells of uniform size, enclosed by bands of fibrous tissue and intersected by finer strands of the latter.

Fig 1.

Fig. 2.

Fig. 3.

F. E. Burgess del.

Mintern Bros. Chromo lith.

Lympho sarcoma of tongue.

Congenital unilateral Hypertrophy of tongue.

T.E. Burgess del. Mintorn Bros. Chromo lith.

Fig. 1.

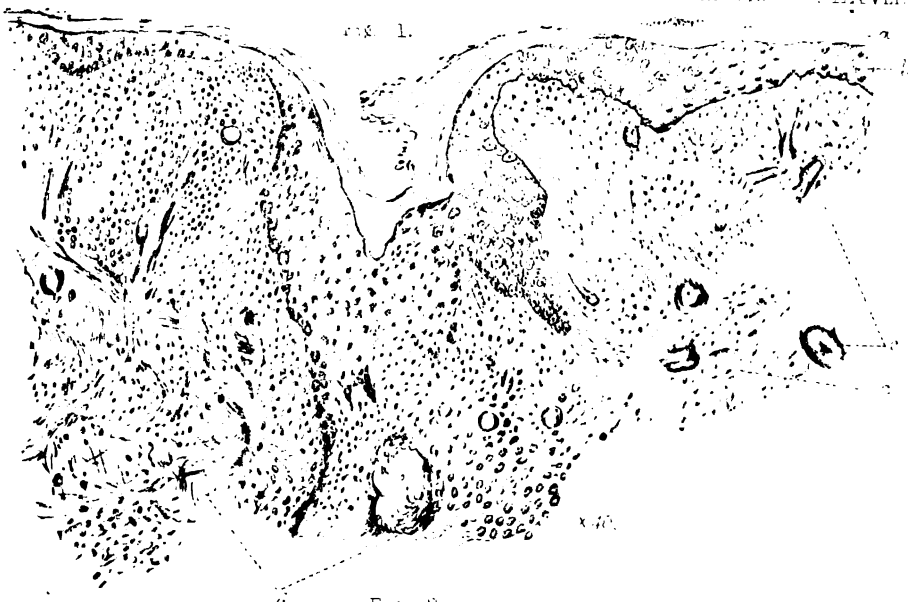
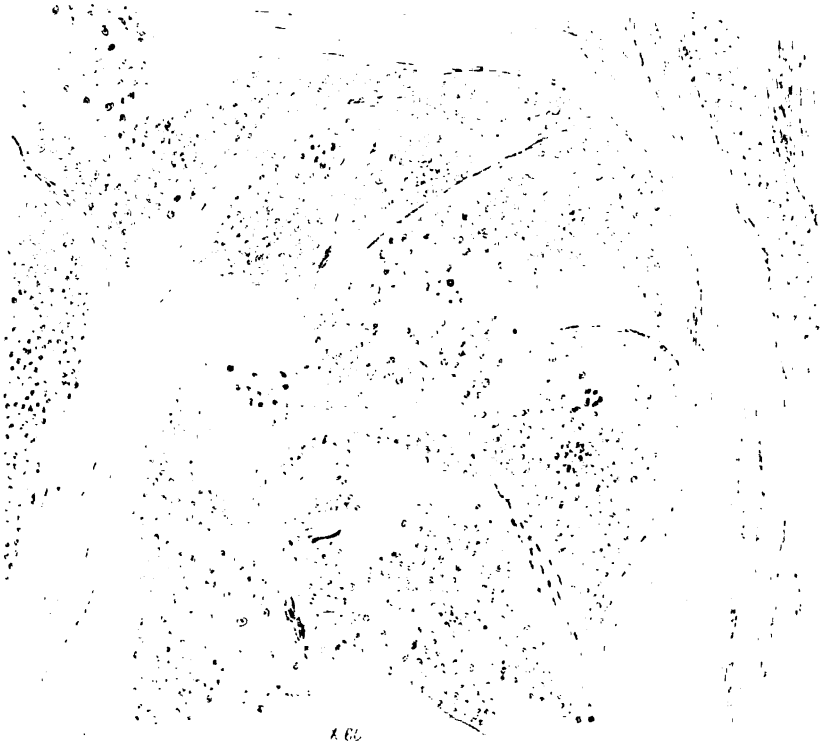


Fig. 2.



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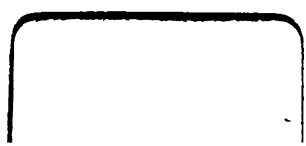
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